STUDY OF PERINATAL OUTCOME
IN OLIGOHYDRAMNIOS IN TERM PREGNANCY

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BONAFIDE CERTIFICATE

This is to certify that this dissertation entitled "**STUDY OF PERINATAL OUTCOME IN OLIGOHYDRAMNIOS IN TERM PREGNANCY**" is the bonafide work done by **Dr.ROSALIND.T**, at the department of Obstetrics and Gynaecology, Government Theni Medical College and Hospital, Theni during her post graduate study for MS Branch II Obstetrics and Gynaecology (2014-2016) from August 2014- July 2015. This dissertation submitted to Dr. MGR Medical University in partial fulfillment of the University rules and regulations for the award of MS Degree in Obstetrics and Gynaecology.

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DECLARATION

I solemnly declare that the dissertation titled, **"Study of Perinatal outcome in Oligohydramnios in term Pregnancy"**, has been prepared by me.

This is submitted to the Tamilnadu Dr. M.G.R. Medical University - Chennai, in partial fulfilment of the requirements for the award of M.S. Degree Examination (Obstetrics and Gynaecology) to be held in April 2016. This record of work has not been submitted previously by me for the award of any degree or diploma from any other university.

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CONTENTS

INTRODUCTION

INTRODUCTION

The amniotic fluid is the fluid that collects within the amniotic cavity surrounding the embryo. It is elaborated by amnion; a two layered extra embryonic membrane formed by inner ectoderm and outer somatic mesoderm.

Source of Amniotic fluid:

Amniotic fluid is an ultrafiltrate of maternal plasma. By the beginning of the second trimester, the amniotic fluid volume becomes an extension of the fetal extracellular space which diffuses through the fetal skin and is similar to fetal plasma. The main source of amniotic fluid is fetal urination. The human fetal urine production appears to be 1 litre to 2 litres per day at term. Another important source is fetal lungs which produces fluid that exits respiratory tract and enters amniotic compartment.

Removal of amniotic fluid:

Fetal swallowing is the main mechanism by which amniotic fluid is removed. however that does not remove the entire amniotic fluid and other unidentified mechanisms must occur.

Volume of amniotic fluid:

The volume of amniotic fluid increases rapidly with the growth of the products of conception averaging about 50ml at 12 weeks of pregnancy. At 20 weeks its volume is about 400ml and it reaches 1000ml at 36 weeks. During the last few weeks of pregnancy its volume decreases about 600-800ml. At 43 weeks, the range varies from 100-600ml.

Role of amniotic fluid during pregnancy:

- It helps keep the baby warm and provides lubrication that keeps them from growing together. In oligohydramnios fingers and toes can become webbed as a result of not enough amniotic fluid circulating in the uterus.
- Amniotic fluid also lets the baby move easily so he can exercise his muscles and strengthen his bones before he's born.
- It acts like a liquid shock absorber for the baby by distributing any force that may push on the mother's uterus.
- It provides Physical space for fetal movement which is necessary for musculoskeletal development.
- It permits fetal swallowing essential for GIT development
- It helps for fetal lung development
- It guards against umbilical cord compression and protects the fetus from trauma.

With amniotic fluid index of <5cm, incidence of oligohydramnios after 34 weeks was 2.3%. Umbilical cord compression during labour is common with oligohydramnios which increases the risk for caesarean delivery for fetal distress and 5 minute APGAR score <7. **(Chauhan 2007)**[1]

The decrease of amniotic fluid volume is associated with stillbirth, increased labour induction, meconium aspiration syndrome, non-reassuring fetal heart pattern and neonatal death. **(Casey & coworkers, 2000)**[2]

This present study is undertaken to assess the perinatal outcome in oligohydramnios (AFI≤5) in term pregnancy.

AIM OF THE STUDY

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To determine the

Perinatal outcome

in Oligohydramnios

in term pregnancies

with AFI≤5cm.

REVIEW OF

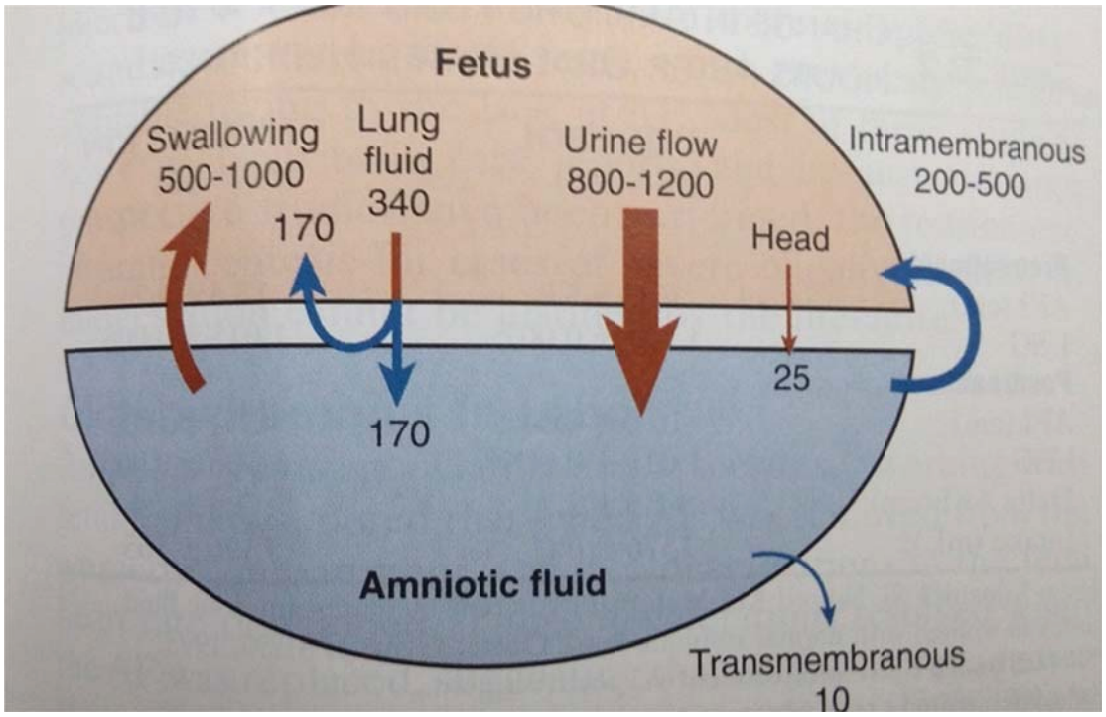
LITERATURE

REVIEW OF LITERATURE

Amniotic fluid:

Amniotic fluid has number of important roles in embryo and fetal development. It provides several important functions to the fetus. The important roles of amniotic fluid are:

- Amniotic fluid volume maintains amniotic fluid pressure thereby reducing the loss of lung liquid, an essential component to lung development. **(Nicolini, 1989)**[3]
- Permitting the movement of fetus and musculoskeletal development.
- Swallowing the fluid leading to gastrointestinal development.
- Protect fetus from trauma
- Prevent cord compression
- Has bacteriostatic properties and prevents infection
- Maintains fetal body temperature
- Provide nutrition to fetus



Pathways of movement of amniotic fluid

6 proposed pathways for fluid movement into and out of amniotic cavity **(Brace, 1997)**[4]

Pathways	ml/day to fetus	ml/day to amniotic fluid
Swallowing by the fetus	500-1000	-
Oral secretions	-	25
Respiratory tract secretions	170	170
Fetal urine	-	800-1200
Intramembranous flow between placeta, umbilical cords and fetus	400	200-500
Transmembranous flow from amniotic cavity into uterine circulation	-	10

Formation of amniotic fluid:

Formation in first trimester is from embryo's plasma volume, which rapidly diffuses across the permeable embryonic skin into amniotic cavity.

In embryo-fetal development, the excretory system develops from pronephros, mesonephros and metanephros. Metanephros begins to develop by 7 weeks of gestational age, and becomes functional by 10-11 weeks.

The glomerular filtration precedes tubular function, so fetal urine is relatively hypotonic initially. As the fetus matures, resorption of sodium, chloride and water occur and the excretion of urea and creatinine increases. **(Mannie IW, 1980)**[5]

Fetal skin becomes cornified and stratified in second trimester, which decreases the diffusion of fluid into amniotic cavity.

Fetal urine is the main source of amniotic fluid. Fetal kidney starts to make urine before the end of first trimester. Urine production increases until term. Fetal urine production rate at 36 weeks of gestation is 230 ml per day which increase to 655ml per day at term. Fetal urine is the major contributor to the volume of amniotic fluid in the latter half of pregnancy. It has been estimated

that the volume of urine produced per day during the latter half of gestation is 35% of its body weight. **(Hedriana, 1994)**[6]

Fetal swallowing starts during early gestation and contributes to the major amniotic fluid removal.Abramovich injected colloidal gold into the amniotic compartment and found that fetal swallowing increases with advanced gestational age. The fetal swallowing of amniotic fluid is evidenced by presence of epidermal debris including lanugo hair in the meconium. It has been estimated that the fetus swallows amniotic fluid which is equivalent to 15% of its body weight. **(Pritchard, 1965)**[7]

Intramembranous pathway of exchange occurs on fetal surface of placenta between amniotic fluid and fetal blood. In this, water and solutes move in opposite directions. It has been estimated that 400ml of water is absorbed intramembranously from the amniotic fluid daily in latter half of gestation.

Fetal lung secretes fluid, of which <1% is used for growth and expansion of fetal lungs. The rest enters into amniotic cavity or is swallowed into trachea. This tracheal fluid contains surfactants which is used as an indicator for lung maturity of the fetus. Fetal lungs absorb fluid during distress only. This is shown by the fact that, meconium staining of amniotic fluid is common,

aspiration of meconium into lungs of the newborn is relatively uncommon. Lung secretion rates are approximately 10% of fetal body weight. **(Harding R 1994)**[8]

Fetal oral and nasal secretions also enter the amniotic fluid, but it has been found to be less than 1% of the body weight per day. **(Brace R.A, 1994)**[9]

Transmembrane pathway of exchange within uterine wall between amniotic fluid and maternal blood. It is as little as 10ml daily near term under normal conditions. **(Brace, 1995)**[10]

INTRAMEMBRANOUS ABSORPTION

There is a discrepancy of 500-750ml /day amniotic fluid by actual proposed mechanism of amniotic fluid formation and removal. Fetal swallowing could not remove entire amniotic fluid volume entering the amniotic compartment from the fetal urine production and lung liquid. This discrepancy can be explained by intramembranous pathway. This process explains the movement of water and solutes between the amniotic compartment and fetal blood that circulates through placental fetal surface. This intramembranous absorption accounts for 200 to 500 ml per day of amniotic fluid removal.

VOLUME REGULATION OF AMNIOTIC FLUID:

In third trimester when the amniotic fluid is around 700-800ml with a daily turnover of 1000ml. Minor or moderate aberration over a period of weeks can result in oligohydramnios or polyhydramnios.

A control loop maintains the AFV at normal by regulating the fetal urine flow, lung liquid secretion, swallowing. Intramembranous absorption is regulated by intramembranous permeability and surface area.

Slight changes in intramembranous permeability can have very large effects on intramembranous flux rates. Any substance eg: prostaglandins excreted by fetal kidneys or released by amnion or chorion which enter the amniotic fluid could potentially alter intramembranous permeability and thus lead to ateration in AFV **(Brace R.A, 1997)**[4]

CHANGES IN AFV ACROSS GESTATION:

AFV changes in pregnancy were studied by Brace and Wolf 1989 and their observations were:

- AFV rises progressively during gestation until 32 weeks.
- From 32 weeks to term, mean AFV is realtively constant (700-800ml)
- After 40 weeks progressive decline in AFV at a rate 8% per week, with amniotic fluid volume averaging only 400ml at 42 weeks.

OLIGOHYDRAMNIOS:

DEFINITION:

Oligohydramnios is defined as decrease in the amniotic fluid level below normal. (Normal level 8-15cm) It is associated with congenital anomalies and perinatal mortality. Oligohydramnios can result in subjective crowding of the fetus in utero.

Oligohydramnios can occur in 1-2% of pregnancies approximately**. (Casey, 2000)**[1] (Petrozole, 2011)

When no measurable fluid pocket is seen, it is referred as anhydramnios.

The pathophysiology of oligohydramnios before membrane rupture is unclear. The theory is the reduced perfusion of the placenta causes hypovolemia, and an

automatic redistribution of fetal blood volume to vital organs resulting in reduced blood supply to kidneys leading to reduced production of urine**.**

TECHNIQUES	DEFINITION	REDELLENCE
Dye dilution	200ml	**Horsager et al (1994)**[11]
Dye dilution	500ml	**Magann et al (1992)**[12]
Direct measurement	318ml	**Brace and Wolf (1989)**[13]
Ultrasound	Single vertical pocket <0.5cm	**Mercer et al (1984)**[14]
Ultrasound	Single vertical pocket <1cm	**Manning et al (1981)**[15]
Ultrsound	Single vertical pocket <2cm	**Manning et al (1990)**[16]
Ultrasound	Single vertical pocket <3cm	**Crawley et al (1984)**[17]
Ultrasound	Two diameter pocket (vertical * horizontal) <15cm	**Magann et al (1992)**[12]
Ultrasound	AFI <5th percentile for GA	**Moore (1990)**[18]
Ultrasound	AFI <5cm	**Phelan (1987)**[19]
Ultrasound	AFI <7cm	**Dizon-Townson (1996)**[20]
Ultrasound	AFI <8cm	**Jeng et al (1992)**[21]

Borderline AFI or borderline oligohydramnios is controversial. AFI between 5-8 is called as borderline oligohydramnios by some. (Baron, 1995)[22] (Magann, 2011) (Petrozella, 2011)

ASSESSMENT OF OLIGOHYDRAMNIOS:

INVASIVE AND NON INVASIVE METHODS:

Amniotic fluid volume can be assessed by both invasive and noninvasive tests. Invasive tests like indicator dilution technique are accurate but tough for clinical use. So we prefer the noninvasive sonographic assessment. The advantage of this method is that it can be done serially for follow up. The assessment of amniotic fluid volume could be subjective or by specific ultrasound measurements. Subjective assessment depends on the experience of the examiner, and reported as adequate, average, scanty.

A single criterion cannot be considered better or superior than others. But using AFI over single deepest pocket assessment can help in identifying more pregnancies with oligohydramnios. But there is no evidence of improvement of pregnancy outcome. **(Nabhan, 2008)**[23]

SONOGRAPHIC ASSESSMENT:

Semiquantitative assessment of amniotic fluid can be done by assessing the amniotic fluid pocket, amniotic fluid index (AFI), and amniotic fluid distribution.

SINGLE POCKET ASSESSMENT:

Chamberlain et al (1984)[24] defined a normal maximal vertical pocket as 2 to 8cm. Measurements below 2cm were called as oligohydramnios and those above 8cm were hydramnios.

With normal maximal vertical pocket (MVP), the perinatal mortality is 2-4/1000. With decreasing amniotic fluid volume (MVP) of 1-2cm it increases 13 fold, and with MVP (<1cm) the mortality increases to 47 fold.

Manning and platt (1981)[15] measured the single deepest pocket of amniotic fluid free of fetal extremities and umbilical cord to assess amniotic fluid volume. This was redefined as normal amniotic fluid as one pocket that measures atleast 2cm in two perpendicular planes. (Manning, 1995)

Halperin et al and Crowley et al (1984)[17] defined 3cm as the limiting value between normal and oligohydramnios. This is found to be a better cut off than 2cm in predicting adverse perinatal outcome.

In case of twin pregnancies, with twin twin transfusion syndrome, oligohydramnios can be defined as a single deepest pocket having amniotic fluid measurement ≤ 2cm. (Society for Maternal and fetal medicine, 2013)

TWO DIAMETER POCKET:

Magann et al (1992)[12] calculated amniotic fluid volume by multiplying vertical depth of MVP by its largest horizontal diameter. Oligohydramnios is defined as 0 to 15cm^2, Normal as 15 to 50cm^2, hydramnios as more than 50cm^2**.**

FOUR QUADRANTS AMNIOTIC FLUID POCKETS:

Phelan, (1987)[19] assessed amniotic fluid as summation of maximum vertical pocket of amniotic fluid in each of the four quadrants of uterus. Each pocket should have ≥5mm width. He defined normal as 8.1 to 18cm, low as 5.1 to 8 cm, very low as ≤5cm, high as >18cm. In condition were AFI <10cm, it is preferable to use mean of three AFI measurements.

With 15 – 24 weeks gestational age, AFI is calculated as summation of MVP in two halves of uterus only.

Abdominal pressure exerted by transducer can also cause changes in AFI. Low pressure results in 13% increase in amniotic fluid index, while high pressure could lead to 21% amniotic fluid decrease.

The diagnosis may also be based on Moore nomogram as a AFI below 5th percentile for gestational age. In this 50th percentile of amniotic fluid index as 12.4cm in term pregnancy, and 5th, 10th, 90th, 95th percentile as 8.1, 9.0, 13.5,

14.4cm. With 28-42 weeks gestation, values below 5th percentile serve as the lower limit of normal amniotic fluid index. According to normative data of **Moore and Cayle (1990)**[25] AFI below 1st and 5th percentile will have more adverse outcomes as compared to between 5th and 95th percentile.

Petrozella and colleagues, 2011 found that with gestational age 24-34 weeks, and AFI 5-8cm were not more likely to be complicated by maternal hypertension, stillbirth, neonatal death as compared to those with AFI more than 8cm.

Oligohydramnios is usually taken as AFI ≤ 5cm or a single deepest pocket of amniotic fluid ≤ 2cm (ACOG, 2012)

Reliability of amniotic fluid volume assessment by ultrasonogram:

Ultrasound assessment of amniotic fluid is a semiquantitative menthod.so there is a question of reliability. Normal volume are best identified by this method whereas decreased /increased liquor are not accurately identified. In addition to this ,other practical difficulties can occur like inexperienced operator, fetal position, chances of transient change in amniotic fluid volume and the different criteria for abnormal amniotic fluid volume. Furthermore there is no accurate cut off for predicting morbidity and mortality of fetus. With low or normal amniotic fluid volume observer variation of 1.0 -2.0 cm is observed. With excess amniotic fluid, 3 fold greater observer variation is found.

Comparison of AFI and Single deepest pocket:

There are studies comparing amniotic fluid index and single deepest pocket giving conflicting results. According to Moses et al, neither the amniotic fluid index nor 2×1 pocket technique as admission test, identifies a pregnancy at risk for an adverse perinatal outcome. In singleton uncomplicated postterm pregnancies, the number of abnormal AFI was significantly higher than the abnormal maximum pool depths. There was increasing trend towards ceasarean section particularly for fetal distress. Morris et al, found AFI less than 5 but not a single deepest pocket less than 2 cm was associated with birth asphyxia, caesarean section and low apgar scores. These studies conclude that amniotic fluid index is superior to single deepest pocket in identifying at risk fetus.

AMNIOTIC FLUID DISTRIBUTION:

It is calculated by comparing the sum of amniotic fluid in lower quadrant fluid pockets to upper quadrant fluid pockets. This should be equal.

CAUSES OF OLIGOHYDRAMNIOS:

Decrease in amniotic fluid volume in second or third trimester is likely to be associated with fetal growth restriction, placental abnormality or maternal preeclampsia. The underlying etiology is uteroplacental insufficiency which can impair fetal growth and reduce fetal urine output.

FETAL:

- Chromosomal abnormalities
- Congenital anomalies
- Growth restriction
- Fetal demise
- Post term pregnancy
- Ruptured membranes

PLACENTAL:

- Abruptio placenta
- Twin to twin transfusion

MATERNAL:

- Hypertension
- Preeclampsia
- Diabetes
- Uteroplacental insufficiency
- Hypovolemia

IATROGENIC:

- Prostaglandin synthethase inhibitors
- NSAIDs: Fetal ductus arteriosus constriction, decresed fetal urine production
- ACE inhibitors

  ↓

  fetal hypotension

  ↓

  renal hypoperfusion

  ↓

  Renal ischemia

  ↓

  anuria, renal failure

  ↓

  Decreased amniotic fluid
- Chorionic villous sampling

IDIOPATHIC:

FETAL ANOMALIES: (Mccurdy and Seeds, 1993)[26] (Peipert and Donnenfeid, 1991)[27]

- Amniotic band syndrome
    - CNS: Holoprosencephaly, Meningocele, encephalocele, microcephaly
- CVS: TOF, septal defects
- Cloacal dysgenesis
- Chromosomal abnormalities – triploidy, trisomy 18, turners
- Cystic hygroma
- Diaphragmatic hernia
- Genitourinary: Renal agenesis, Renal dysplasia, urethral obstruction, Potter syndrome, Meckel Gruber syndrome, Prune belly syndrome
- Hypothyroidism
- Twin to twin transfusion
- Twin reversed arterial perfusion sequence
- Skeletal: Sirenomelia, Sacral agenesis, Absent radius, facial clefting
- VACTERL anomaly (vertebral, Anal, Cardiac, Tracheo-esophageal, Renal, Limb)

Fetal kidneys are major contributor of amniotic fluid volume by 18 weeks of gestational age. So among all anomalies, most severe oligohydramnios are associated with genitourinary anomalies.

Trimmer and coworkers (1990) sonographically measured hourly urine production by finding sequential bladder measurements in pregnancies of ≥ 42 weeks. Oligohydramnios was found to be associated with diminished urine production.

So when there is bilateral renal agenesis, no urine can be produced, and this will result in oligohydramnios or even anhydramnios. Due to lack of amniotic fluid, fetus can develop limb contractures, compressed face, or even death due to pulmonary hypoplasia. This set of anomalies occurring due to bilateral renal agenesis, is called potter syndrome after Dr. Edith Potter, who described it in 1946.When this set of anomalies occur associated with some other etiology causing decreased amniotic fluid volume, it is called potter sequence.

During middle of second trimester if amniotic fluid cannot be visualized, because of some genitourinary etiology, it will result in extremely poor prognosis. Fetal therapy is an option in these circumstances.

Aneuploidies and other genetic syndromes cause oligohydramnios indirectly either from fetal decompensation, fetal growth restriction or accompanying placental abnormality. The prevalence of congenital anomalies and aneuploidy varies between 4.5-37% and 0.4-4% respectively. **(Nicolaides, 1991)**[28] (Shipp, 1996)

Assessing fetal anatomy is difficult with reduced amniotic fluid volume. So transvaginal sonography, and colour or power Doppler can be used to confirm the presence of kidneys and renal arteries. **(De Vore, 1995)**[29]

Early symmetric intrauterine growth restriction and oligohydramnios suggests a possible karyotypic abnormality. **(Nicolaides, 1991)**[28]

POTTERS FACIES

IUGR BABY

INTRA UTERINE GROWTH RESTRICTION AND OLIGOHYDRAMNIOS:

IUGR results in oligohydramnios due to decreased urine production secondary to decreased uteroplacental perfusion. Recent studies show the cause to reversal of intramembranous flow . When single pocket of amniotic fluid is >2cm, between 1&2cm, <1cm, prevalence of IUGR is %, 20%, and 37%. **(Chamberlain, 1984)**[24]

Petrozella and associates (2011) similarly reported that AFI<5cm between 24-34 weeks is associated with high risk for still birth, preterm birth, heart rate abnormalities, and growth restriction.

PRETERM RUPTURE OF MEMBRANES AND OLIGOHYDRAMNIOS:

Rupture of membranes prior to 37 weeks of gestation is called as premature rupture of membranes. It has an incidence of 1.7% between 24-34 weeks of gestation. Survival in such second trimester oligohydramnios is approximately 10%. (Shipp, 1996)

POST TERM PREGNANCIES AND IUGR:

Prolonged pregnancies (>42 wks) leads to diminished placental function and oligohydramnios**. (Elliot 1961)**[30]

Oz and associates (2002) used Doppler waveform and found that fetal renal blood flow is reduced in post term pregnancies complicated by oligohydramnios.

FETAL HYPOXIA AND OLIGOHYDRAMNIOS:

In maternal diseases like chronic hypertension, severe pre eclampsia, connective tissue disorders, chronic renal disease fatal hypoxia occurs due to uteroplacental insufficiency. **(Deutinger, 1987)**[31]

Experimental hypoxia results in a reflex redistribution of fetal cardiac output, a decrease in renal and pulmonary flow, hence urinary output and production of fluid by lung decreases and the amount of amniotic fluid declines.

But under long term conditions, hypoxia can induce suppression of fetal swallowing resulting in increase in AFV. Oligohydramnios in fetal hypoxia is caused by placental dysfunction in addition to hypoxia.

MATERNAL HYPOVOLEMIA AND OLIGOHYDRAMNIOS:

Acute maternal hypovolemia has been found to be the cause of oligohydramnios. **(Sherer, 1990)**[32] The changes in amniotic fluid volume maybe mediated by the changes in intramembranous flow because the water induced reduction in fetal osmolality would be expected to reduce intramembranous absorption. **(Flack, 1995)**[33]

FETAL EFFECTS OF OLIGOHYDRAMNIOS:

In severe early onset oligohydramnios, as in renal agenesis, there are several problems and the fetal outcome is poor. (Shenker & colleagues, 1991) **(Garmel & coworkers, 1997)**[34] These problems may not be seen in late onset oligohydramnios which accompany intrauterine growth restriction. They are

- Pulmonary hypoplasia
- Amniotic adhesions or bands causing deformities like amputation of digits
- Limb deformities like talipes
- Potters facies (Low set ears, epicanthic fold, receding mandible, flattened nose)

Incidence of pulmonary hypoplasia is higher with oligohydramnios **(Mossinger & colleagues & Winn & associates, 2000)**[35]**.** According to **Fox and Badalian (1994)**[36] and **Laura and colleagues (1995)**[37]**,** there are three possibilities that account for pulmonary hypoplasia.

1. Thoracic compression prevents chest wall excursion and lung expansion
2. Lack of breathing movements decreases lung inflow
3. Failure to retain amniotic fluid leading to impaired lung growth and development

Third trimester oligohydramnios causes malpresentation, umbilical cord compression, concentration of meconium in liquor, difficult or external cephalic version. **(Hofmeyr, 1991)**[38]

Baron and colleagues (1995)[39] reported 50% increase in variable decelerations during labour and seven fold increase in cesarean delivery.

Sarno and coworkers (1989-1990)[40] reported that AFI ≤ 5 was associated with fivefold increase in cesarean delivery rates.

Casey and coworkers showed a 25% increase in non-reassuring fetal heart rate pattern when women with oligohydramnios were compared with normal controls. However the cesarean rate for this increased only from 3 to 5%. Intrapartum complications:

1. Cord compression in labour causing variable derceleration
2. Meconium aspiration syndrome

FETAL DISTRESS AND OLIGOHYDRAMNIOS:

Leveno and colleagues (1984) described the risks to post term fetuses. Antepartum and intrapartum fetal distress were found to be a consequence of cord compression due to oligohydramnios. The volume of amniotic fluid

decreases after 38 weeks and passing meconium into a reduced amniotic fluid results in thick viscous meconium which may be swallowed by the fetus resulting in meconium aspiration syndrome.

OLIGOHYDRAMNIOS AND NON REACTIVE NST:

Spontaneous deceleration in no stress test with AFI<5cm may predict fetal compromise. Hoskein et al showed in a study of 3150 patients of >34 weeks, that fetuses with antepartum decelerations had statistically significant increased incidences of intrapartum distress regardless of AFI. They also had significantly increased rates of neonatal acidosis and low APGAR scores when there were severe decelerations and AFI≤5cm in the antepartum period.

The incidence of clinical oligohydramnios and NST revealing fetal heart decelerations or bradycardia and an increase in the association with neonatal acidosis and low APGAR scores was found to increase as the sonographic estimates of amniotic fluid volume were decreased. So it is suggested that the postdated pregnancy with evidence of reduced AFV should be considered for a trial of labpour with cardiotocogram continuously. In contrast an AFI>5cm coupled with normal NST has been correlated with a low incidence of fetal death (<1/1000) within a week.

MANAGEMENT OF OLIGOHYDRAMNIOS:

Managing oligohydramnios requires proper assessment of mother and fetus to find the underlying aetiology and correct it so the amniotic fluid returns back to normal. Eg. Discontinuing prostaglandin inhibitor

Intruterine growth restriction needs testing, and optimal time for delivery.

In preterm premature rupture of membranes, need to administer antibiotic and corticosteroids till gestational age of 32 weeks. (Vermillion, 2000)

Isolated third trimester oligohydramnios may not always be associated with poor outcome for the baby. **(Magann, 1999)**[41]

AMNIOINFUSION:

In women with oligohydramnios, amnioinfusion can be tried prophylactically to avoid intrapartum fetal heart rate abnormalities due to cord compressions. Nageotte and coworkers found that this significantly decreased frequency and severity of variable deceleration.

In a randomized trial, Macri and colleagues, studied prophylactic amnioinfusion in 170 term and post term pregnancies complicated by both thick meconium and oligohydramnios. Amnioinfusion significantly reduced the cesarean delivery rates for fetal distress and meconium aspiration syndrome.

TRANSABDOMINAL AMNIOINFUSION:

Transabdominal amnioinfusion can be tried for diagnostic and therapeutic purpose for second trimester oligohydramnios. **(Quetel, 1992)**[42]

400-600ml Normal saline instilled transabdominally resulted in better ultrasound visualization. Adding indigo carmine and detecting it vaginally, helped identifying preterm premature rupture of membranes. **(Fisk, 1991)**[43]**. Miyazaki and Nevaserz (1985)**[44] noted that various deceleration due to cord compression are reduced by transabdominal amnioinfusion**.**

TRANSVAGINAL AMNIOINFUSION:

Predominant indications are:

- Meconium stained amniotic fluid
- Variable deceleration
- Prophylactically for oligohydramnios

500-800ml bolus of warmed normal saline followed by a continuous infusion of approximately 3ml/min (Owen,1990) (Pressman, 1998)

In another study Rinechart and colleagues (2000) randomly gave a 500ml bolus of NS at room temperature alone or 500ml bolus plus continuous infusion of 3ml/min.

AMNIOINFUSION IN VARIABLE DECELERATION:

Hoffmeyer and Laurie (2012) used the Cochrane Database to specifically analyse the effects of amnioinfusion in management of variable deceleration in fetal heart rate patterns associated with umbilical cord compression. They used 19 studies, most of them having less than 200 subjects. The conclusion was that amnioinfusion was useful in reducing occurrence of variable deceleration, reducing caesarean deliveries incidence, improving neonatal outcome. The American College of Obstertricians and Gynaecologists (2013) recommends using amnioinfusion in cases with persistent variable deceleration.

AMNIOINFUSION FOR MECONIUM STAINED AMNIOTIC FLUID:

Pierce and associates (2000) analysed 13 prospective trials regarding intrapartum amnioinfusion in 1924 women with moderate to thick meconium stained amniotic fluid. They found that infants of mothers treated with amnioinfusion had lesser chance of having meconium below the vocal cords and less likely to have meconium aspiration syndrome as compared to infants of mothers who were not given amnioinfusion. The caesarean incidence was also significantly lower in mothers treated with amnioinfusion. Rathore and colleagues (2002) also found similar results in a separate study.

But Fraser and colleagues (2005) did amnioinfusion in women having thick meconium stained amniotic fluid in labour and concluded it has no significant benefits.

Because of these findings, ACOG (2012) does not recommend amnioinfusion to dilute meconium stained amniotic fluid.

According to Xu and colleagues (2007) where continuous monitoring is not possible, amnioinfusion may be used to lower incidence of meconium aspiration syndrome.

COMPLICATIONS OF AMNIOINFUSION:

- Uterine hypertension
- Chorioamnionitis
- Cord prolapse
- Abnormal fetal heart tracing
- Uterine rupture
- Maternal cardiac or respiratory compromise
- Placental abruption

MATERNAL HYDRATION:

Intravenous hydration with 6500ml of an isotonic increased amniotic fluid volume in markedly dehydrated women. (Sherer, 1990)

Brace (1989)[13] showed that changes in maternal intravascular volume can alter fetal urine output, amniotic fluid volume and to a lesser extent intravascular volume.

Kilpatric and coworkers (1991)[45] oral maternal hydration with two litres of water was associated with an increase in AFI by approximately 30% in women with decreased AFI and normal AFI.

MATERIALS AND METHODS

MATERIALS AND METHODS

Present study is a hospital based study on perinatal outcome in term gestation with AFI ≤ 5cm and control group AFI > 5cm, was carried out in Department of Obstetrics & Gynaecology, Government Theni Medical College Hospital, Theni, during the period of August 2014 – July 2015 Ethical clearance was obtained for this study from the institution.

INCLUSION CRITERIA:

- Singleton pregnancy with Gestational age >37 weeks
- Pregnancies without anomaly with intact membranes
- AFI ≤ 5

EXCLUSION CRITERIA:

- Singleton pregnancy with gestational age <37weeks
- Patients with multiple gestation
- Patients with fetus having congenital anomamlies like renal agenesis, polycystic kidney disease
- Ruptured membranes or draining PV
- Polyhydramnios

SAMPLE SIZE:

Study was conducted to observe outcome of labour in form of perinatal morbidity and maternal outcome in form of vaginal or cesarean section.

Study group: About 150 cases in AFI ≤ 5 cms

Control group: 150 cases with AFI > 5cm

History about the patient's age, obstetric code, gestational age, menstrual history, obstetric history, associated complicationsin present pregnancy were noted. Symphysio-fundal height was measured in centimeters. Fetal movements and fetal heart rates was recorded serially. Blood investigations – hemoglobin, blood grouping and typing, cell counts, blood sugar, urine analysis, HIV, VDRL, USG, Doppler, NST were done. Speculum and per vaginal examination was done to rule out draining per vaginum and confirmed intact membranes. After taking informed consent patients were treated. Iron, calcium, and multi vitamin supplements were continued orally as before. AFI measurements was done. These women were followed till discharge.

Decision of delivery by vaginal route or elective/ emergency LSCS was done as required. Some patients were already in labour and others allowed to go into spontaneous labour. If delivery is made by caesarean section, the indication was recorded.

A pre designed study proforma was filled for each case.

OUTCOME:

The outcome measures were

1. CTG changes
2. mode of delivery
3. presence of meconium
4. APGAR score at 5 minutes

Primary outcome:

Fetal distress as defined by any one or more of the following criteria.

- Recurrent variable deceleration
- Late deceleration
- Prolonged bradycardia
- APGAR score ≤ 6 at both 1 and 5 minutes.

Secondary outcome:

- Mode of delivery – instrumental or cesarean section for fetal distress
- Meconium staining of amniotic fluid
- Need for amnio infusion
- NICU admission

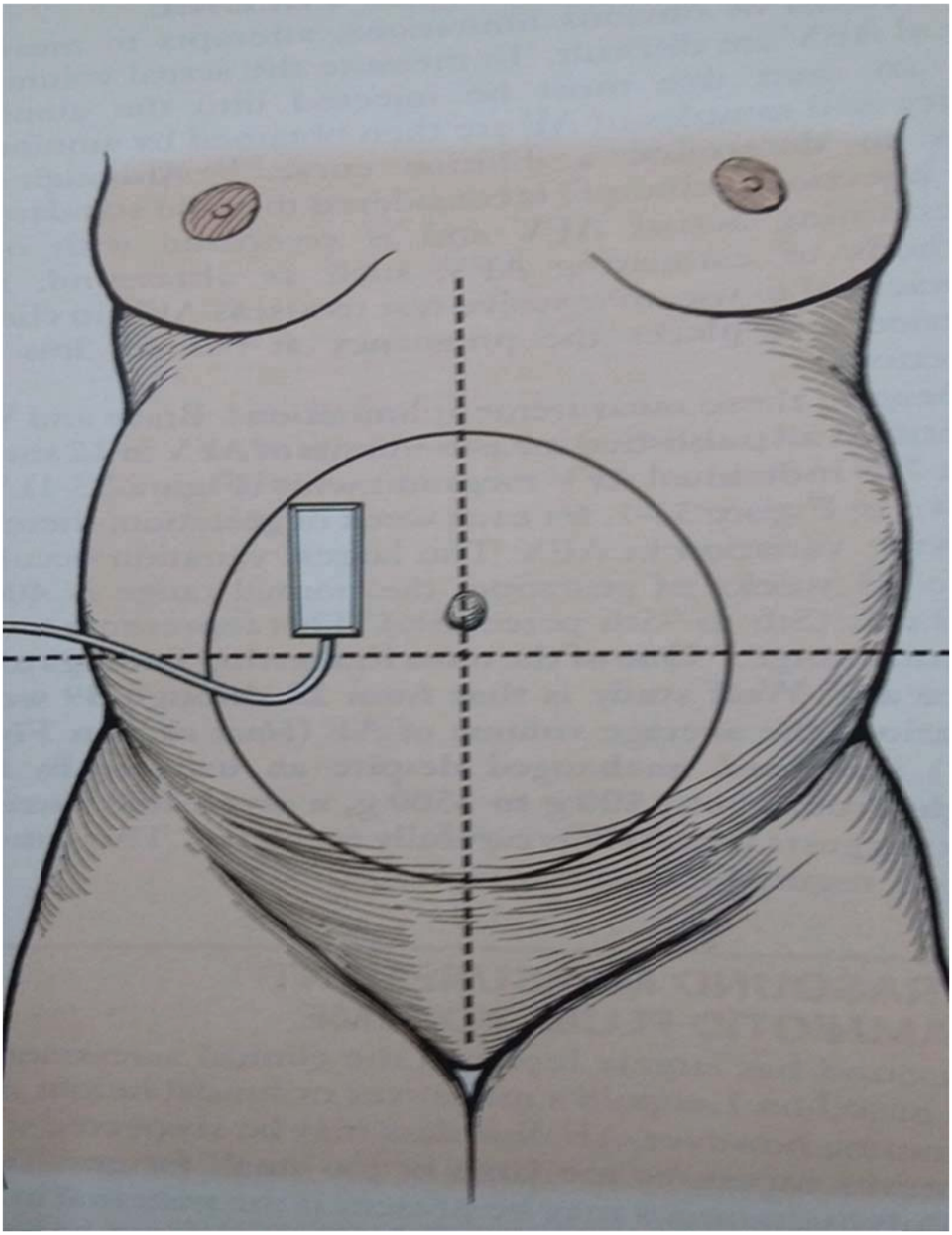
METHODS:

An ultrasound examination was done to monitor fetal wellbeing and assess amniotic fluid index and it was measured by Phelan's technique.

A curvilinear transducer was used. The uterus was divided into four equal quadrants – the right and left upper and lower quadrants respectively through the maternal midline vertically and an arbitrary transverse line between symphysis pubis and upper edge of uterine fundus.

Transducer placement was parallel to maternal sagittal plane and perpendicular to maternal coronal plane.

Image frozen at the clear deepest pocket of amniotic fluid. This pocket was measured using ultrasound calipers in a vertical direction. It is repeated in each of the four quadrants and summation of the four values gives AFI. Patients are grouped according to their AFI, study group with AFI $\leq$ 5 cm, and control group with AFI > 5 cms.



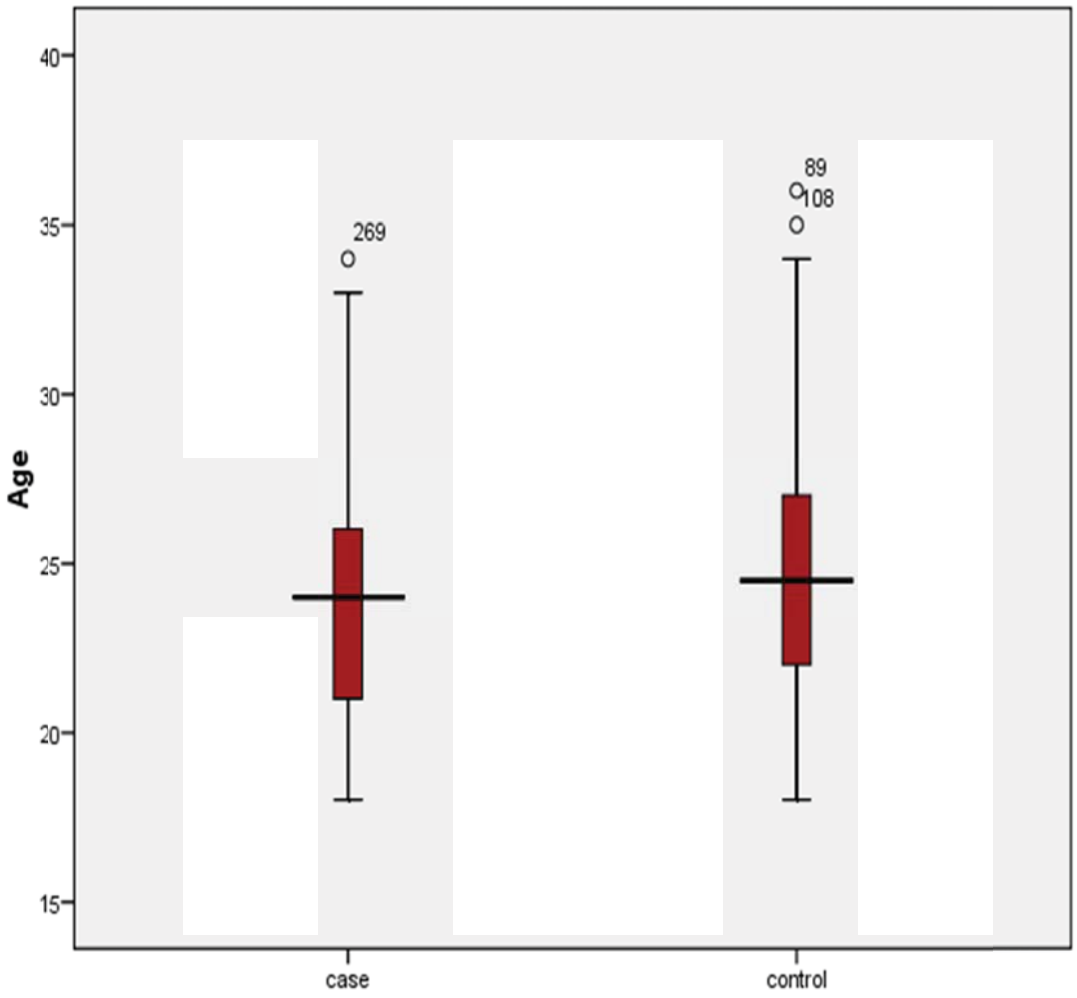
FOUR QUADRANT TECHNIQUE FOR MEASURING AFI

SINGLE DEEPEST POCKET MEASUREMENT

OBSERVATIONS &

RESULTS

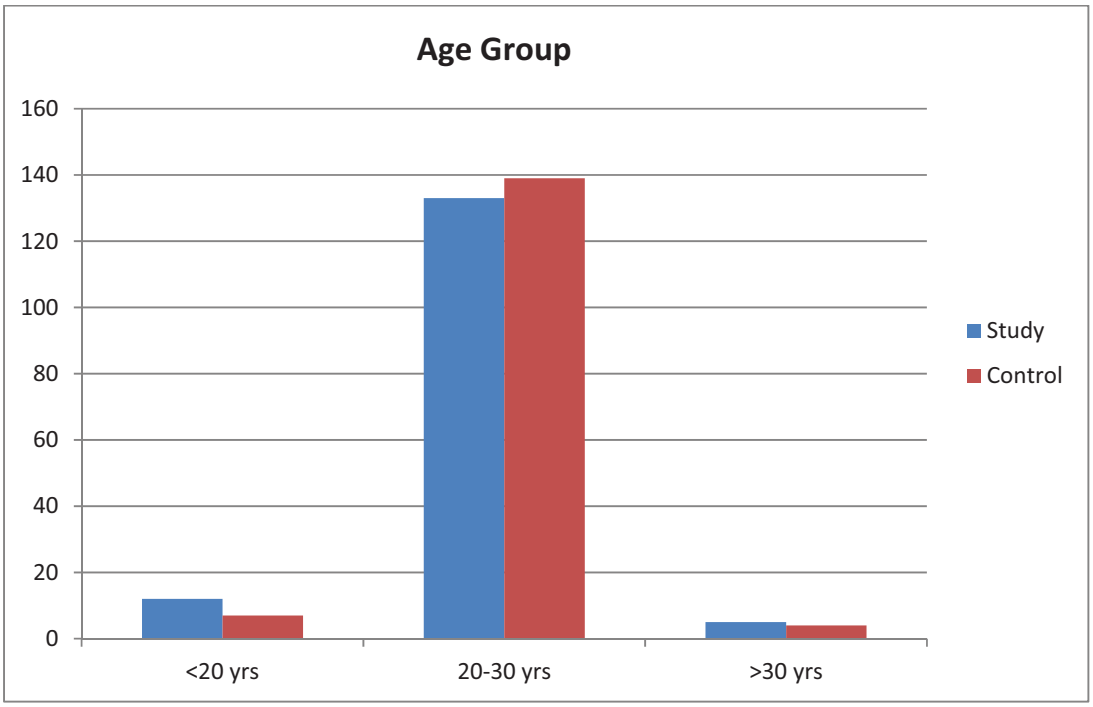
Variable	case	Mean	Std. Deviation	Std. Error Mean	Tstatistic	p
Age	case	23.96	3.227	.263	-2.339	0.020
	control	24.85	3.337	.272		
Gestational Age in weeks	case	38.36	1.485	.121	-0.538	0.591
	control	38.44	1.052	.086		
AFI	case	3.97	.811	.066	-38.502	<0.0001
	control	8.09	1.023	.084		



Box whisker plot of age of study population

Age Group	Study group		Control group	
Age	**No**	**%**	**No**	**%**
<20 yrs	12	8%	7	4.7%
20-30 yrs	132	88%	138	92%
>30 yrs	6	4%	5	3.3%
Total	**150**	100%	**150**	100%

Most patients in this study belong to the age group of 20-30 yrs.


Age Group
160
140
120
100
80
60
40
20
0
<20 yrs
20-30 yrs
>30 yrs
Study
Control

OBSTETRIC CODE				
PARITY	**Study Group**		**Control Group**	
	No	**%**	**No**	**%**
PRIMI	83	55.3%	85	56.7%
MULTI	67	44.7%	65	43.3%

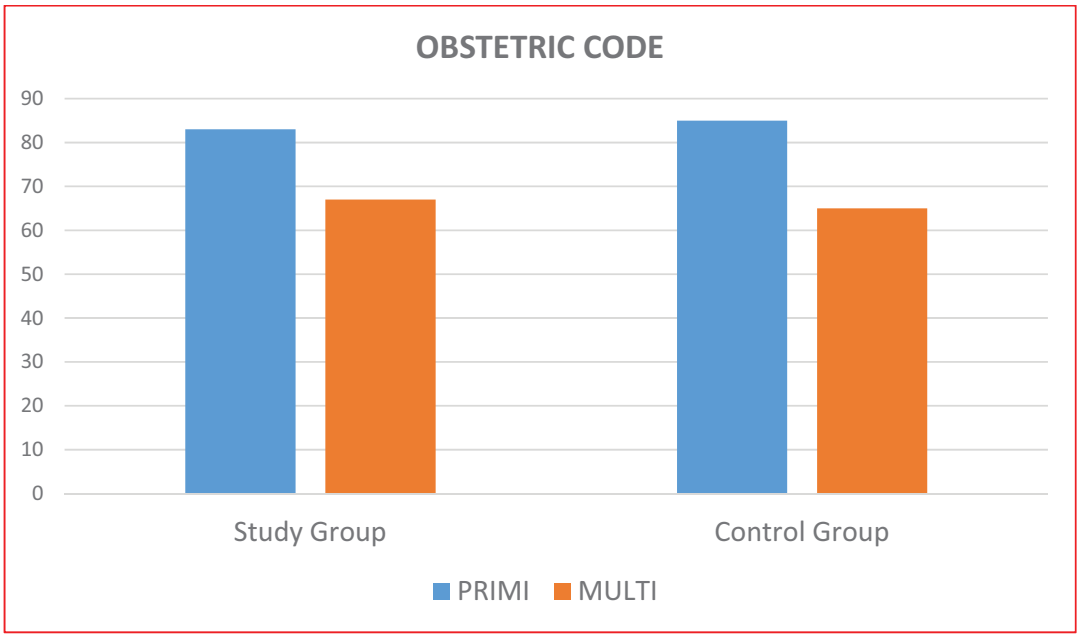
X^2 – 0.054

p-0.81

Most of the patients were primiparous in this study,

55.3% in study group and 56.7% in the control group.

This difference was found to be non-significant.


OBSTETRIC CODE
90
80
70
60
50
40
30
20
10
0
Study Group
Control Group
PRIMI
MULTI

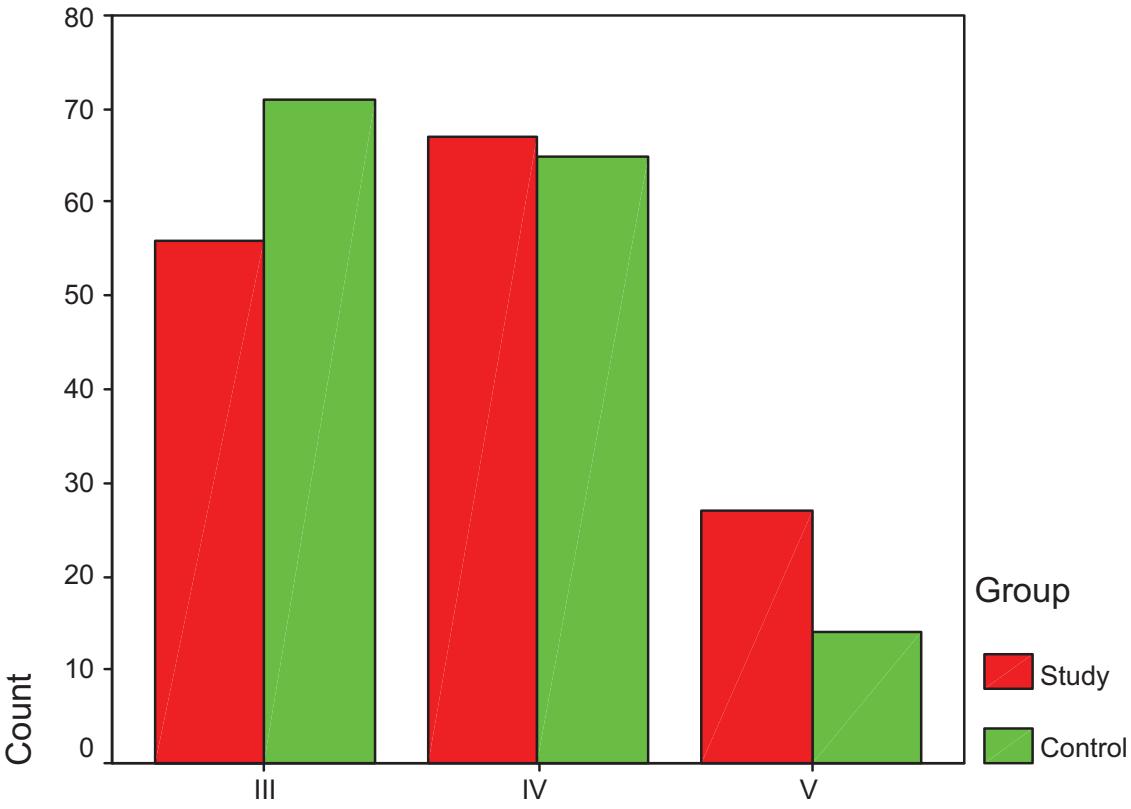
Socioeconomic Status	Study		Control	
	No	%	No	%
III	56	37.3%	71	47.3%
IV	67	44.7%	65	43.3%
V	27	18.0%	14	9.3%

X^2 – 5.94

P – 0.052

Majority of patients in study group were from socioeconomic class IV (44.7%) while in the control group most were from socioeconomic class III (47.3%)

This was also found to be non-significant.



q3g.Socioeconomic status

Gestational age

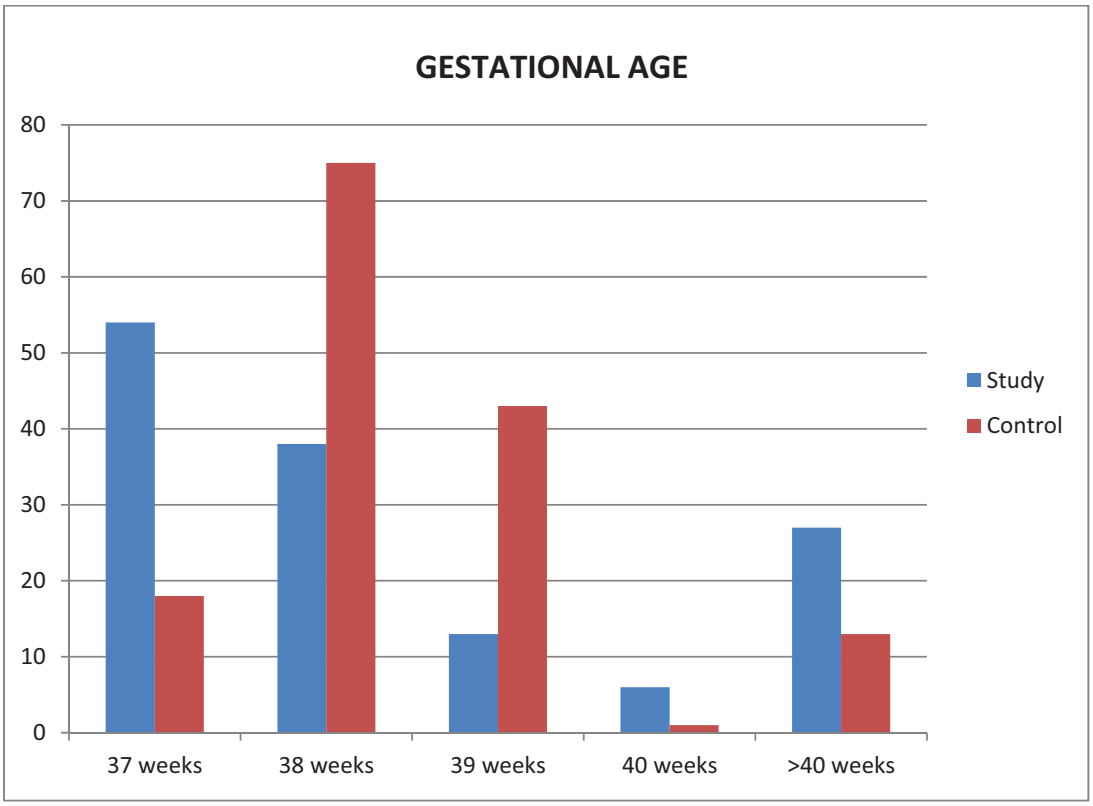
Gestational age	Study		Control	
	No	%	No	%
37 weeks	54	36	18	12
38 weeks	50	33.4	75	50
39 weeks	13	8.6	43	28.7
40 weeks	6	4	1	0.7
>40 weeks	27	18	13	8.6

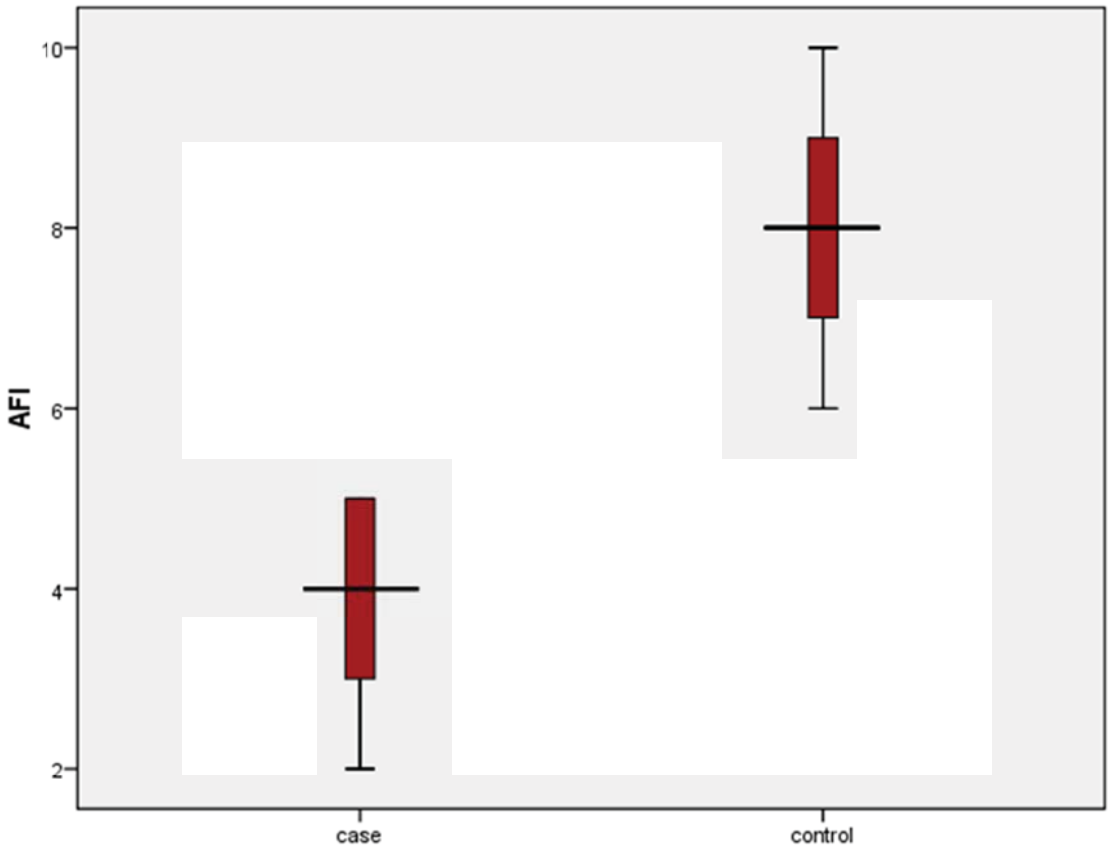
Most pregnancies in study group were delivered by 37 completed weeks. (36%)

In control group, most pregnancies were delivered ny 38 completed weeks. (50%)

Comparatively more pregnancies were continued till term in control group.

The incidence of postdated pregnancies are more in study group. (18%)


GESTATIONAL AGE
80
70
60
50
40
30
20
10
0
37 weeks
38 weeks
39 weeks
40 weeks
>40 weeks
Study
Control



Box whisker plot of AFI of study population

High risk	case	control
Pre eclampsia	26 (17.7)	20(13.6)
Postdated	27(18.4)	13(8.8)
Breech	14(9.5)	3(2.0%)
uncomplicated	56(38.1)	95(64.6)
previous lscs	24(16.3)	19(12.9)

X^2 – 30.337

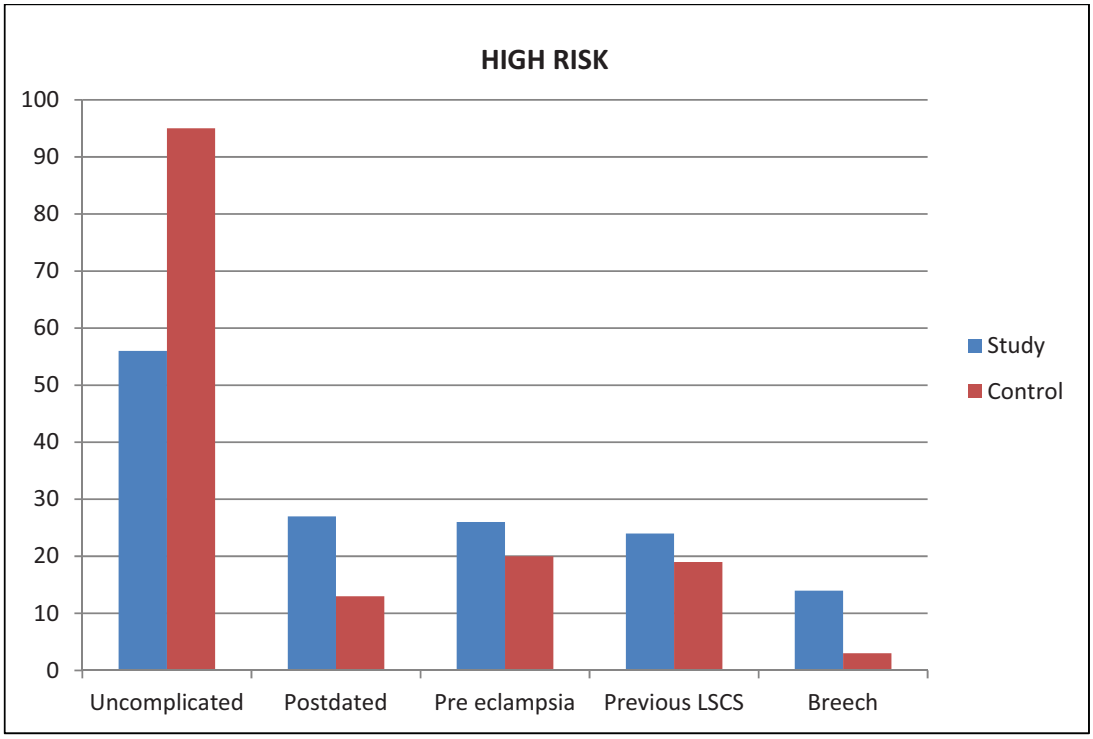
P - <0.0001

Most pregancies were uncomplicated in both study and control group.

In study group, postdated pregnancy was 18.4%, pre eclampsia was 17.7%, previous LSCS was 16.3%.

In control group, pre eclampsia was 13.6%, previous LSCS was 12.9%, Postdated pregnancy was 8.8%.

This difference was significant.


HIGH RISK
100
90
80
70
60
50
40
30
20
10
0
Uncomplicated
Postdated
Pre eclampsia
Previous LSCS
Breech
Study
Control

NST	Study		Control	
	No	%	No	%
R	101	67.3%	125	83.3%
NR	49	32.7%	25	16.7%

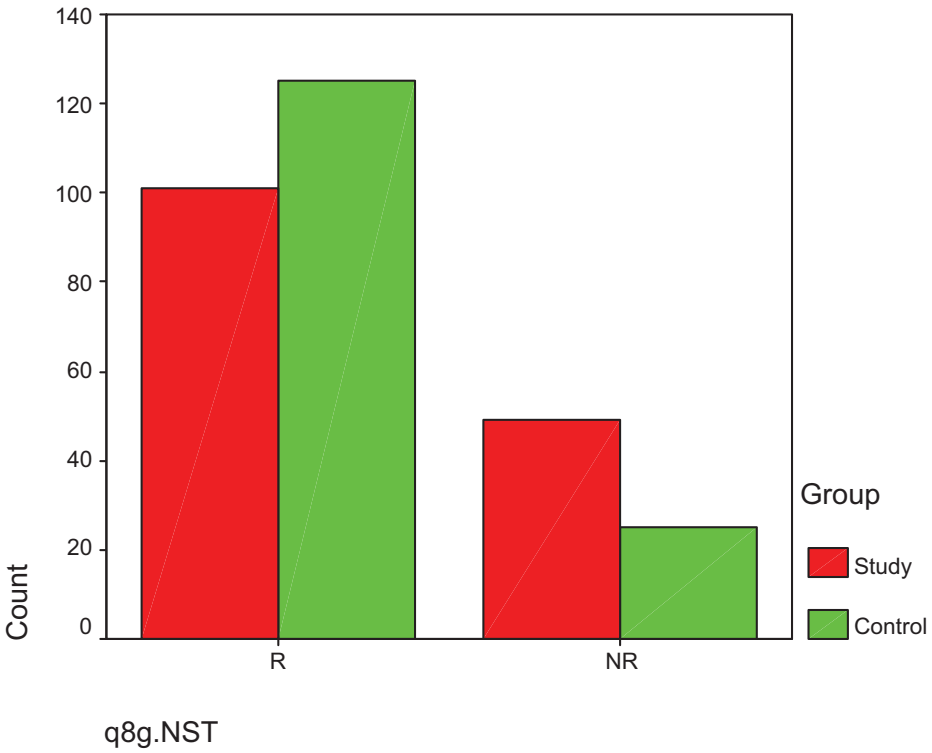
X^2 - 10.332

P – 0.001<0.05

The reactive NST was 83.3% in control group whereas only 67.3% in study group.

The nonreactive NST was 32.7% in the study group as compared to 16.7% in the control group.

This difference was found to be significant.


140
120
100
80
60
40
20
0
Count
R
NR
Group
Study
Control
q8g.NST

Onset of labour	Study		Control	
	No	%	No	%
I	54	36.0%	45	30.0%
S	96	64.0%	105	70.0%

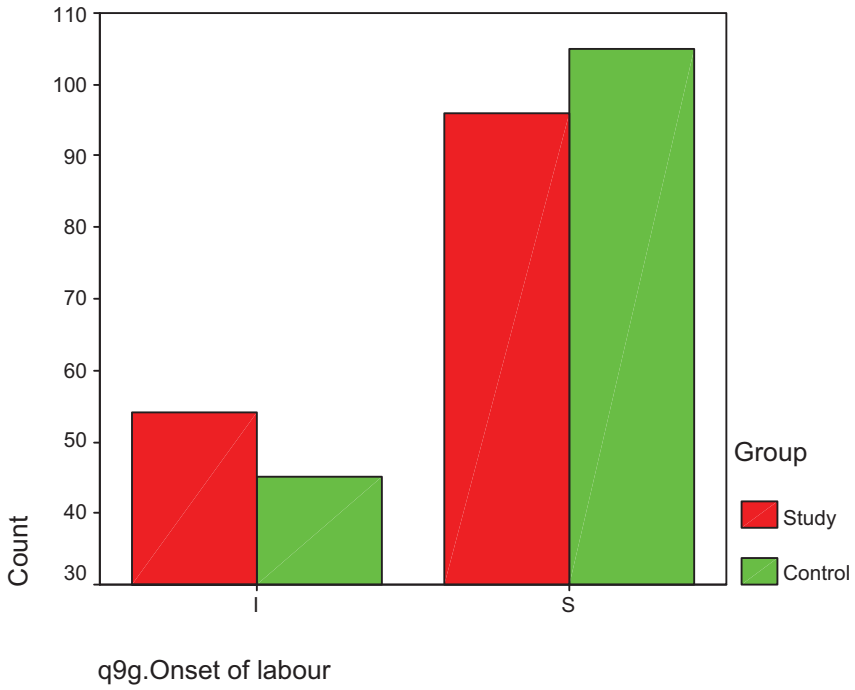
$\mathbf{X}^2$ – 1.221

P - 0.269

Labour occurred spontaneously in 64% of study group and 70% of control group.

Labour was induced in 36% of study group and 30% of control group.

This difference was not found to be significant.


110
100
90
80
70
60
50
40
30
Count
I
S
q9g.Onset of labour
Group
Study
Control

Colour of liquor	Study		Control	
	No	%	No	%
Clear	81	54.0%	110	73.3%
Thick	39	26.0%	11	7.3%
Thin	30	20.0%	29	19.3%

X^2 – 20.100

P – 0.000<0.05

Liquor was clear in 73.3% of patients in control group as against 54% in study group.

It was **thin meconium** stained in

20% of study group and

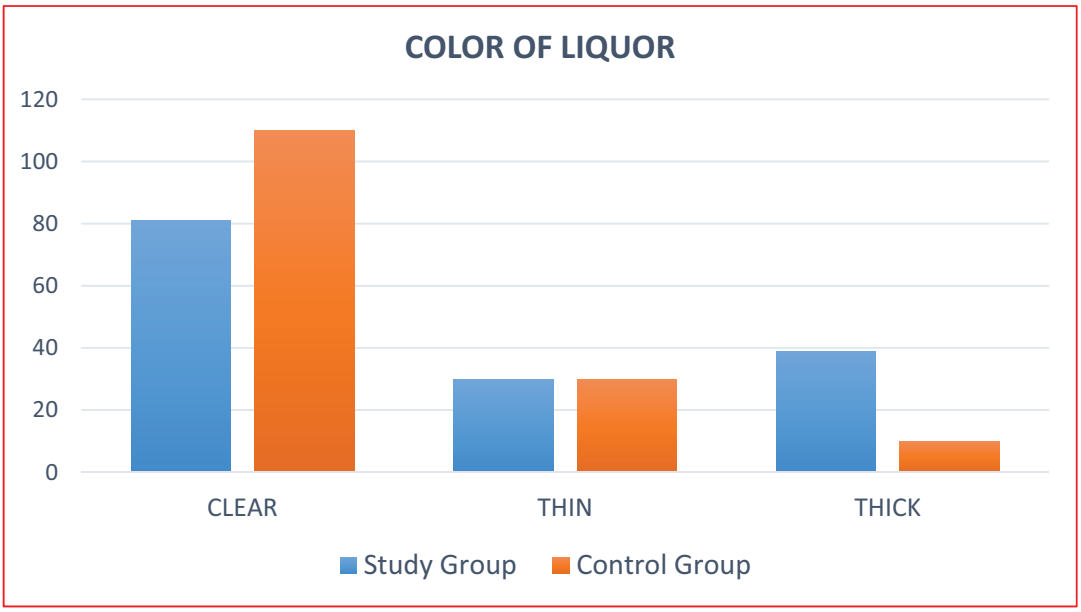
19.3% of control group.

It was **thick meconium** stained in

26% of study group and

only 7.3% of control group.

This difference was found to be significant.


COLOR OF LIQUOR
120
100
80
60
40
20
0
CLEAR
THIN
THICK
Study Group
Control Group

Mode of delivery	Study		Control	
	No	%	No	%
LN	57	38.0%	93	62.0%
LSCS	69	46.0%	35	23.3%
RPT	24	16.0%	22	14.7%

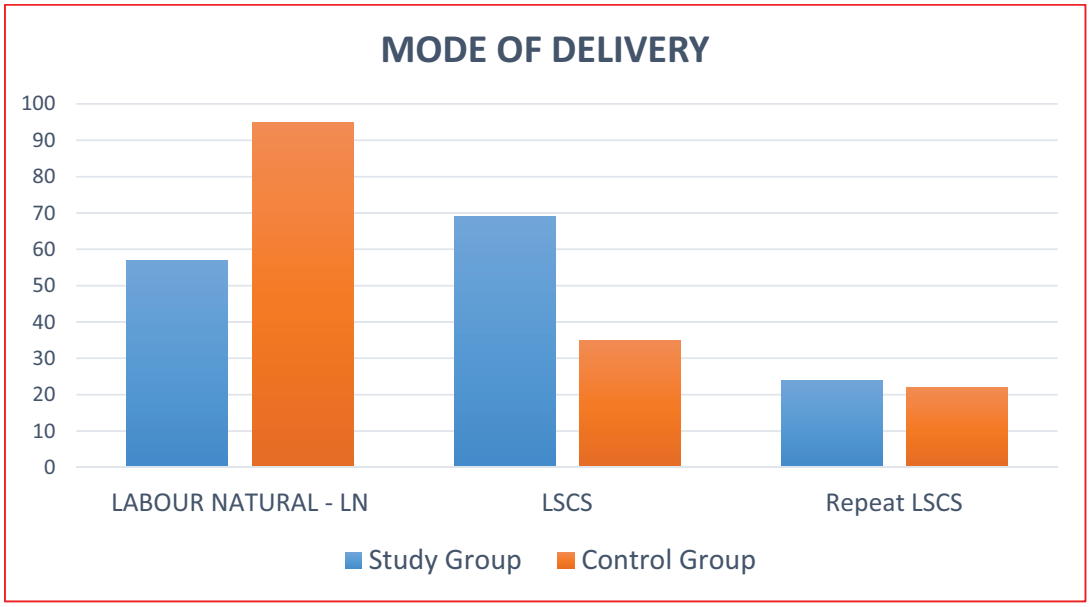
X^2 – 19.842

P – 0.000<0.05

Majority of deliveries in study group were LSCS (46%), with repeat LSCS (16%) and labour natural was only 38%

In control group, labour natural was 62% and LSCS was only 23.3% with repeat LSCS 14.7%

This difference was found to be significant.


MODE OF DELIVERY
100
90
80
70
60
50
40
30
20
10
0
LABOUR NATURAL - LN
LSCS
Repeat LSCS
Study Group
Control Group

Indication for LSCS	Study		Control	
	No	%	No	%
Breech	9	6.0%	3	2.0%
CPD	10	6.7%	19	12.7%
FD	50	33.3%	17	11.3%
FI	13	8.7%	11	7.3%
IUGR	10	6.7%	5	3.3%
Others	2	1.3%	2	1.3%

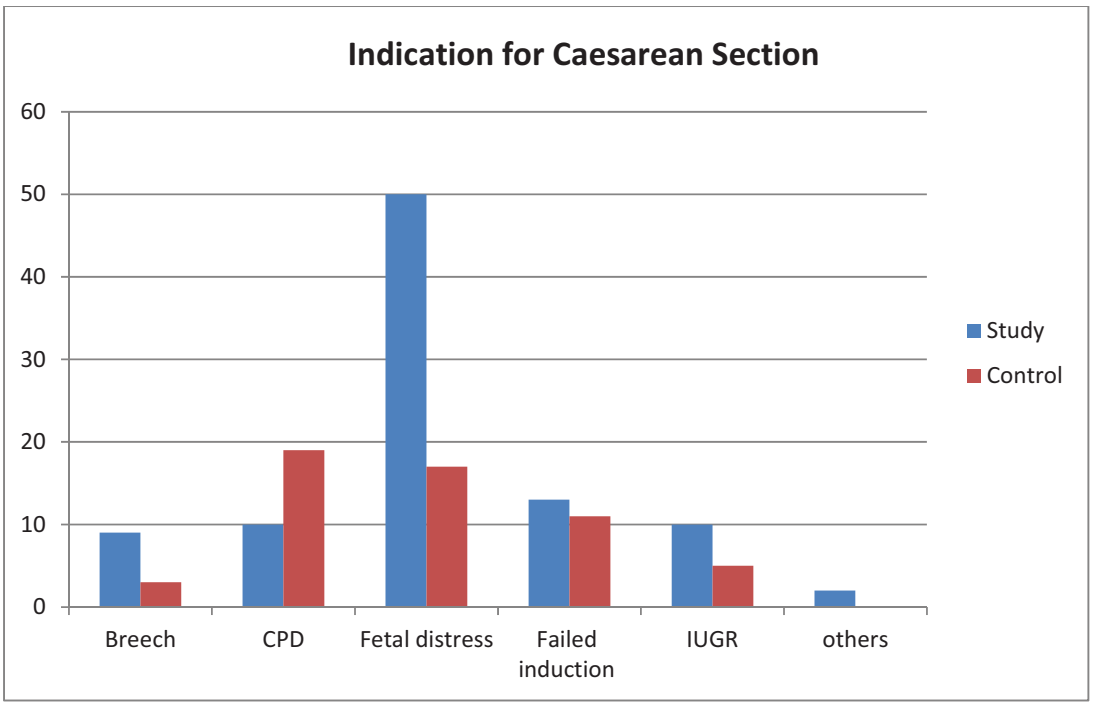
X^2 – 33.068

P – 0.000<0.05

Among study group, the most common indication for LSCS is fetal distress – 33.3%.

In control group, only 11.3% of patients underwent LSCS for fetal distress.

This difference was found to be significant.


Indication for Caesarean Section
60
50
40
30
20
10
0
Breech
CPD
Fetal distress
Failed induction
IUGR
others
Study
Control

Baby weight	Study		Control	
	No	%	No	%
Below 2kg	38	25.3%	12	8.0%
2 to 2.5kg	24	16.0%	27	18.0%
2.5 to 3kg	75	50.0%	83	55.3%
3kg & above	13	8.7%	28	18.7%

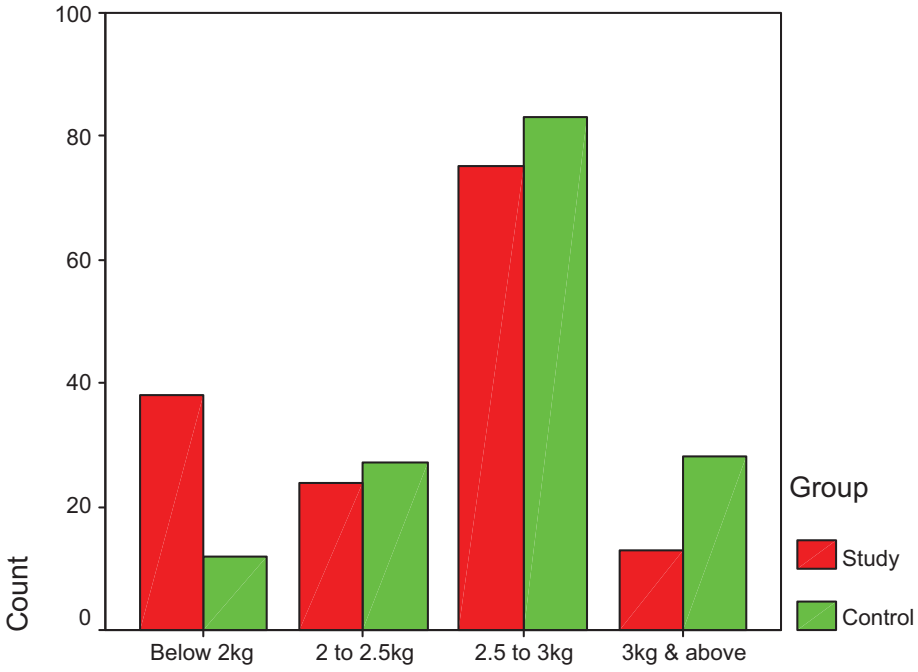
X^2 – 19.589

P – 0.000<0.05

Among the study group 25.3% babies were <2kg,

whereas in control group only 8% babies were <2kg.

This difference was found to be significant.



q13.Baby weight

Apgar	Study		Control	
	No	%	No	%
Below 4	6	4.0%	0	.0%
4 to 7	20	13.3%	3	2.0%
7 & above	124	82.7%	147	98.0%

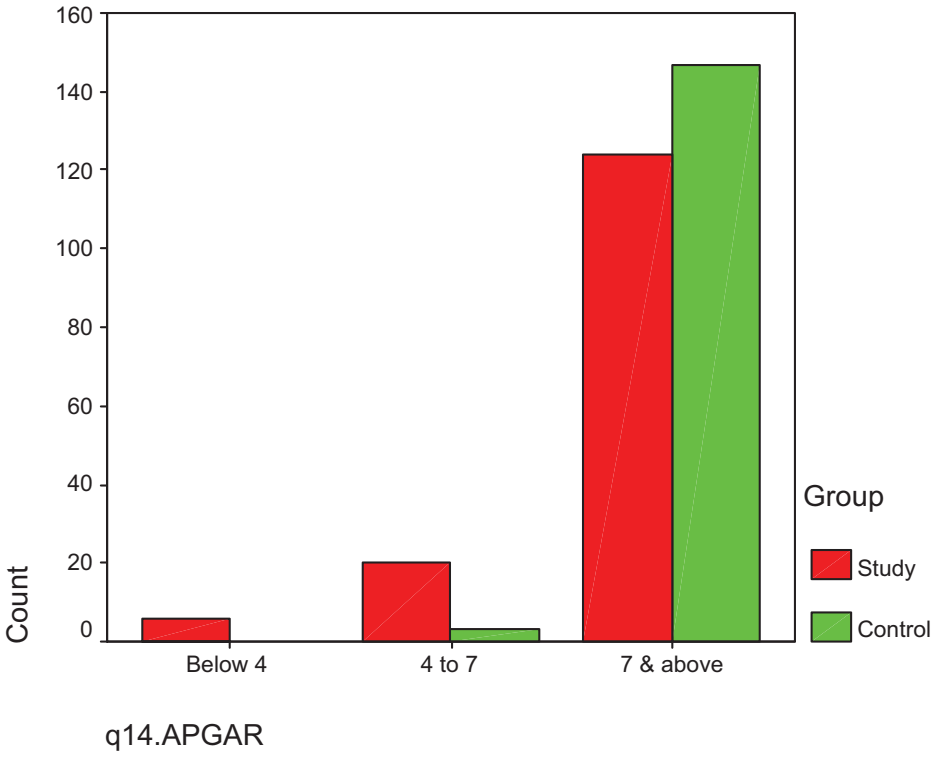
X^2 – 20.517

p-0.000 <0.05

APGAR score was above 7 in 82.7% patients in study group,

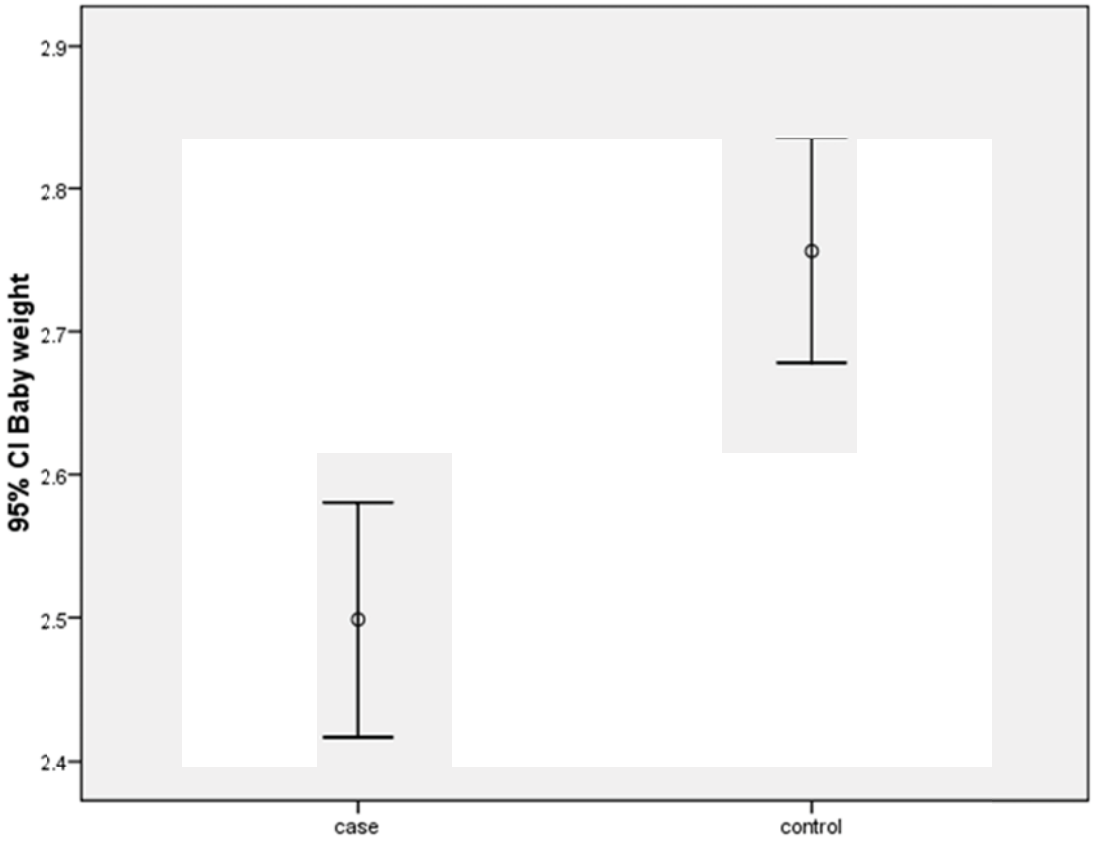
and 98.0% patients in the control group.

This difference was found to be significant.

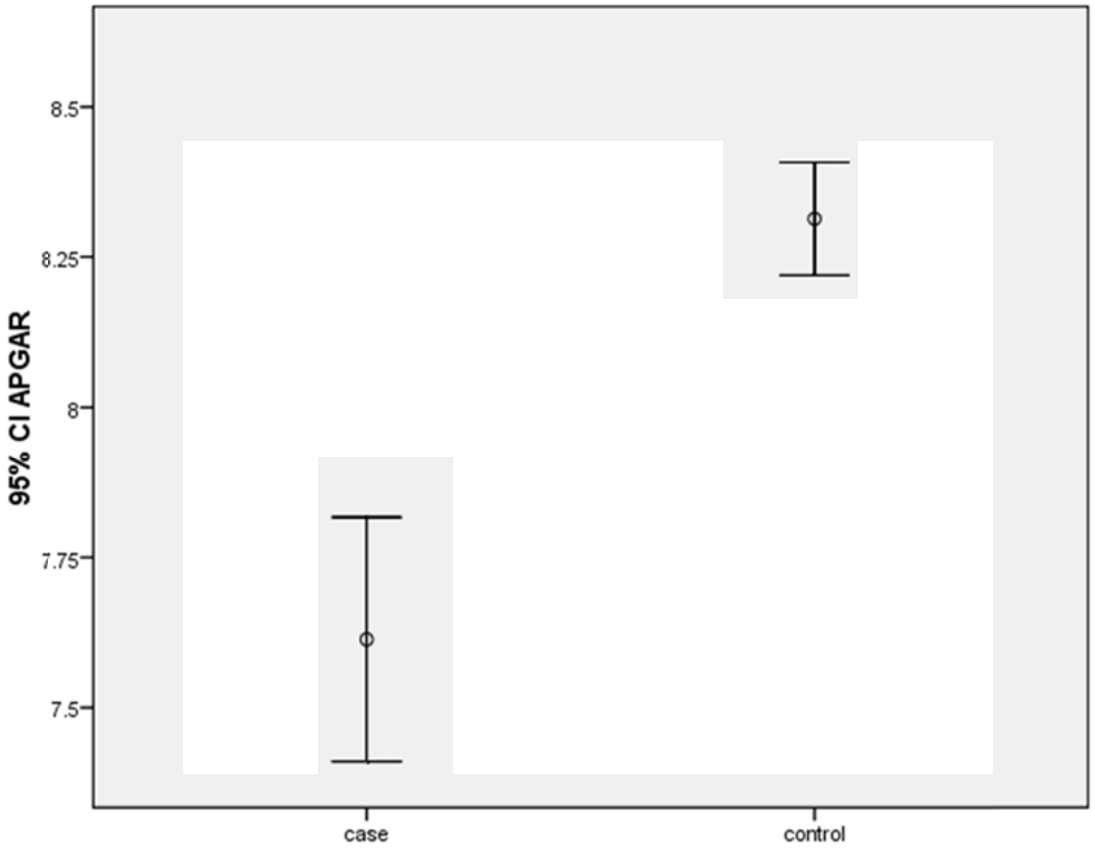

160
140
120
100
80
60
40
20
0
Count
Below 4
4 to 7
7 & above
q14.APGAR
Group
Study
Control

Baby characteristics among study population

Variable	case	N	Mean	Std. Deviation	Tstatistic	p
Baby weight	case	150	2.499	.5065	-4.498	<0.0001
	control	150	2.757	.4869		
APGAR	case	150	7.61	1.257	-6.190	<0.0001
	control	150	8.31	.581		



Error bar diagram showing baby weigh distribution among cases and controls



Error bar diagram showing apgar score distribution among cases and controls

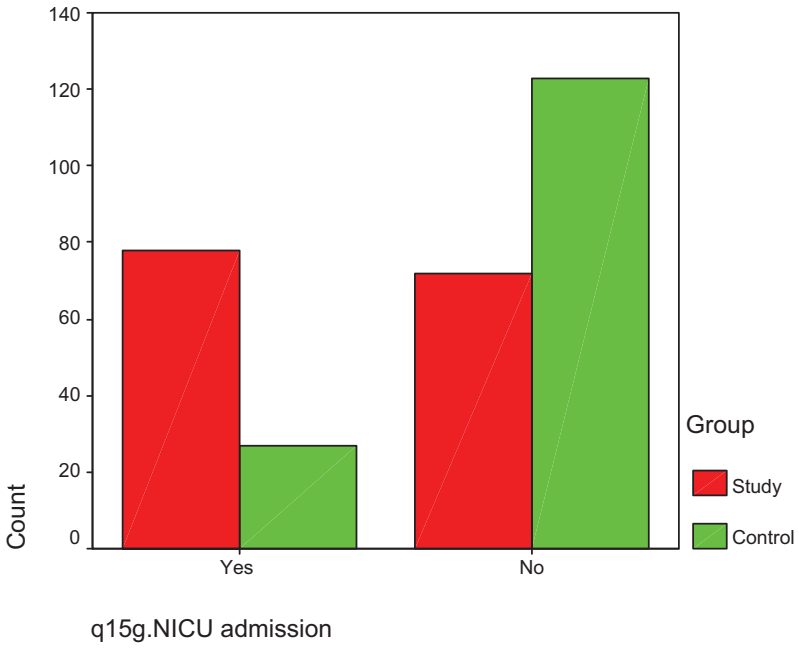
NICU admission	Study		Control	
	No	**%**	**No**	**%**
Yes	78	52.0%	27	18.0%
No	72	48.0%	123	82.0%

X^2 – 38.110

P – 0.000<0.05

52% of babies in study group were admitted in NICU, but only 35% of babies in control group were admitted in NICU.

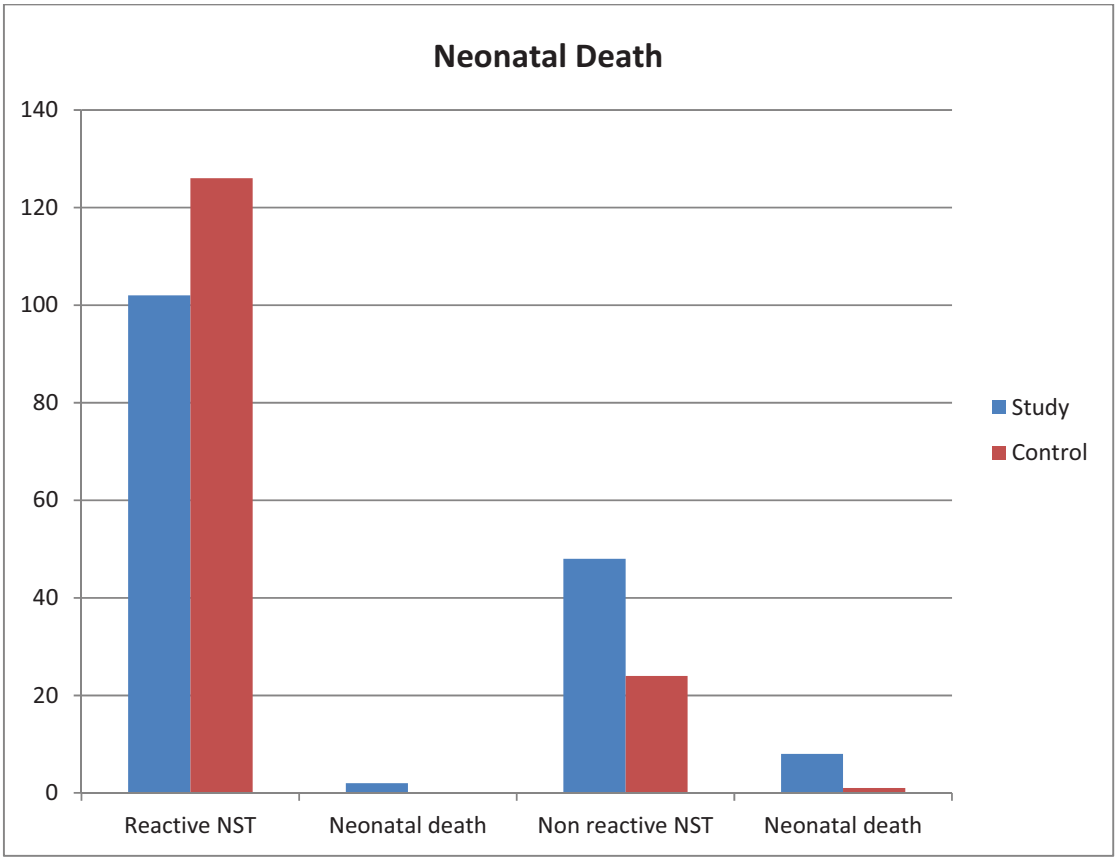
This difference was found to be significant.


140
120
100
80
60
40
20
0
Count
Yes
No
q15g.NICU admission
Group
Study
Control

NEONATAL OUTCOME				
Neonatal Outcome	**Study Group**		**Control Group**	
	No	%	No	%
REACTIVE NST (R-NST)	102	68%	126	84%
NEONATAL DEATH	2	2%	NIL	
NON REACTIVE NST (NR-NST)	48	32%	24	16%
NEONATAL DEATH	8	16%	1	4%

In the study group, 68% babies had reactive NST and 32% babies had non-reactive NST. Among this, neonatal death occurred in 2% of babies with reactive NST, and 16% of babies with non-reactive NST.

In control group, 84% babies had reactive NST and 16% babies had non-reacvtive NST. Among this, there was no death with reactive NST and 4% neonatal death among babies with non-reactive NST.


Neonatal Death
140
120
100
80
60
40
20
0
Reactive NST
Neonatal death
Non reactive NST
Neonatal death
Study
Control

Effects of NST results on baby – study group

Variable	categories	NST		Chi sq	p
		Non reactive	Reactive		
apgarfive	<7	16 (32.7)	10(9.9)	11.919	<0.0001
	>7	33 (67.3)	91(90.1)		
NICU admission	Yes	41(83.7)	37(36.6)	29.249	<0.0001
	No	8(16.3)	64(63.4)		
Final outcome	DISCHARGED	32(65.3)	83(82.2)	14.267	0.003
	DEATH	1(2)	0		
	Death IUGR	7(14.3)	1(1)		
	IUGR	9(18.4)	17(16.8)		

Effects of NST results on baby – control group

Variable	categories	NST		Chi sq	p
		Non reactive	Reactive		
apgarfive	<7	2 (8)	1(0.8)	5.510	0.019
		8.0%	.8%		
	>7	23 (92)	124(99.2)		
		92.0%	99.2%		
NICU admission	Yes	11(44)	16(12.8)	13.740	<0.0001
	NO	14(56)	109(87.2)		
Final outcome	Discharged	21(84)	117(93.6)	0.961	0.327
	IUGR	3(12)	7(4.7)		
	Death	1(4)	0		

Final outcome on study population

Final outcome	case	control
Normal	115 (76.6)	139(92.7)
Death	9(6)	1(0.7)
IUGR	26(17.3)	10(6.6)

X^2 – 17.349

P – 0.001

Neonatal death occurred in 6% of study group and 0.7% of control group.

DISCUSSION

DISCUSSION:

Oligohydramnios with AFI ≤ 5cm can lead to an increase in perinatal mortality and morbidity. Under these conditions, there is increased frequency of meconium stained liquor, fetal distress, low apgar scores, abnormal fetal heart rates.

Compared to control group, there was a two fold increase in neonatal and fetal acidosis. (Moore et al, 1997) There was a threefold increase in caesarean section for fetal distress compared to control.

In this study, we had 150 cases in the study group with AFI ≤ 5cm. And the control group had 150 cases with AFI > 5cm.

In the study group, 26 patients had pre eclampsia, 27 cases had postdated pregnancy, 24 of them had previous LSCS, and 14 cases were with breech presentation.

In control group, 20 patients had pre eclampsia, 13 cases had postdated pregnancy, 19 of them had previous LSCS, and 3 cases were with breech presentation.

Casey & coworkers (2001) conducted a study on pregnancy outcome after diagnosis of oligohydramnios, and found that there was an increase in induction of labour (42% over 18%), nonreassuring fetal heart rate patterns (48% vs

39%), NICU admission (7% over 2%), MSAF (1% over 0.1%), neonatal death rate (5% over 0.3%) associated with oligohydramnios.

Chamberlain & coworkers 1993, found there was a significance between incidence of congenital anomaly , IUGR related to amniotic fluid volume.

Youseef et al 1993 conducted a study on measurement of AFI and fetal outcome and found AFI more than 5cm had better chance of predicting a good fetal outcome.

Golan & coworkers (1994) assessed fetal outcome in 145 babies with oligohydrmanios and found increased incidence of fetal distress, MSAF (29%), IUGR (24.5%), breech (17%), birth asphyxia (11.5%).

Chauhan S P & coworkers (1999) found increased risk of caesarean delivery with antepartum and intrapartum AFI $\leq$ 5cm, due to fetal distress and such babies had low apgar score at 5 mins.

Baron and coworkers (2000) compared patients with AFI $\leq$ 5cm with normal AFI patients. Oligohydramnios resulting in caesarean section due to fetal distress was studied and found to have sensitivity of 78%, specificity of 74%, positive predictive value of 33%, negative predictive value of 95%.

Locatelli A 2004 suggested that oligohydrmanios was associated with high risk of low birth weight in postdated pregnancies.

PRESENT STUDY:

In this study outcome of 150 patients with AFI ≤ 5cm was compared with 150 patients with AFI > 5cm.

In study group, 57 patients had normal vaginal delivery and 93 patients underwent LSCS. (69 primary LSCS and 24 repeat LSCS)

In control group, 93 patients had normal vaginal delivery, and 57 patients underwent caesarean section. (35 primary LSCS and 22 repeat LSCS)

	AFI ≤ 5cm	**AFI >5cm**
Labour natural	57	93
LSCS	93	57

INDUCTION OF LABOUR:

Induction	**AFI ≤ 5cm**	**AFI >5 cm**
Casey and coworkers 2000	42%	18%
Present study	36%	30%

In present study, 54 cases in study group, 45 cases in control group were delivered by inducing labour.

FETAL DISTRESS AS INDICATION FOR CAESEREAN SECTION:

Baron Morgan et al (2000)	78%
Present study	74.6%

Oligohydrmanios as a predictor for caesarean section due to fetal distress has a sensitivity of 74.6% comparable with various studies.

MECONIUM STAINED LIQUOR:

Youseef et al (1993)	63.6%
Present study	63.8%

Sensitivity of meconium stained liquor is 63.8% comparable to earlier studies.

Fetal wellbeing score (APGAR) at 5 mins < 7:

Youseef et al (1993)	88.8%
Present Study	90%

Sensitivity for predicting APGAR value < 7 at 5 mins by oligohydramnios in this study is 90% comparable with earlier studies.

IUGR:

Youseef et al (1993)	79.9%
Present Study	75%

Oligohydramnios predicts occurrence of IUGR babies with a sensitivity of 75% in this study and this is comparable with earlier studies.

PERINATAL MORTALITY:

	AFI ≤ 5cm	**AFI > 5cm**
Casey and coworkers (2000)	5%	0.3%
Present study	6%	0.7%

Neonatal death rate is 6% in study group comparable with earlier studies.

NEONATAL DEATH

In the study group there were 9 neonatal deaths out of this 8 were due to complications of IUGR. There was one neonatal death in control group.

- CASE NO:16

  Pandiselvi, 24 yrs, primi, belonging to class 3 socioeconomic status,GA 40 wks 4days on admission with NR –NST.AFI 4cm with unfavourable cervix.emergency LSCS was done.Indication : Fetal distress with IUGR.Alive girl baby 1.6kg APGAR 7/10,8/10 ,thick meconium admitted in NICU.Baby was term IUGR had non- necrotizing enterocolitis, meningitis and jaundice.The baby died after 8 days.

- CASE N0:22

  Geetha,23 yrs primi belongning to class 3 socioeconomic status.GA 40 wks 6 days on admission with NST –NR.AFI 4 with thick meconium in early labour taken up for emergency LSCS within half an hour.Alive girl baby 1.8 kg APGAR 6/10,7/10 with thick meconium ,IUGR admitted in NICU and died after 3 days due to meconium aspiration syndrome.

- CASE NO:23

  Mangai,27 yrs primi belonging to class 4 socioeconomic status GA 37 wks 3 days with pre eclampsia on admission AFI 4 .Gel induction

done.NST NR.emergency LSCS done in view of fetal distress and pre eclampsia.Alive girl baby 1.9 kg APGAR 6/10,7/10 admitted in NICU and baby died 2 days after admission due to RDS.

- CASE NO:39

  Mallika,21 yrs primi belonging to class 4 socioeconomic status GA 37 wks 3 days on admission with pre eclampsia.NST NR.Alive boy baby 1.6kg.APGAR 7/10,8/10.meconium stained liquor.term IUGR.admitted in NICU.baby had recurrent seizures and meningitis.died after 4 days.

- CASE NO:44

  Chitra ,21 yrs primi belonging to class 4 socioeconomic status GA 37 wks 2 days on admission NST-Reactive.Spontaneous labour.Alive girl baby.wt-1.4kg.APGAR 7/10,8/10.clear liquor.term IUGR.admitted in NICU and died after 4 days due to RDS.

- CASE NO:49

  Ambika,23 yrs primi belonging to class 3 socioeconomic status GA 38 wks 3 days ,breech AFI 3 ,NST-NR,emergency LSCS done.indication – primi breech with IUGR with severe oligohydramnios and fetal distress.Alive boy baby wt.1.7kg APGAR 7/10,8/10 thick meconium term IUGR admitted in NICU and baby died after 3 days due to RDS.

- CASE NO:65

  Subadra ,28 yrs primi belonging to class 5 socioeconomic status GA 38 wks with pre eclampsia.AFI 5 gel induction done.NST NR.emergency LSCS done. Indication primi with fetal distress. Baby wt.1.8kg APGAR 3/10,5/10.clear liquor. admitted in NICU. baby died after 1 day due to RDS.

- CASE NO: 86

  Priya, 21 yrs old primi, belonging to class 3 socioeconomic status, 38 weeks GA on admission with pre eclampisa, had AFI 5. She went in for labour spontaneously and her NST was nonreactive. Liquor was thin meconium stained. She delivered by labour natural. Baby had birth weight 1.7kg and APGAR 3/10, 3/10. Baby was admitted in NICU. Baby had polycythemia and died after 6 hrs due to IUGR and pulmonary hemorrhage.

- CASE NO: 147

  Rani, 26 yr old primi, belonging to class 4 socioeconomic status, 37 weeks GA on admission with fetus in breech presentation and AFI 3. Her NST was nonreactive and liquor was found to be thick meconium stained. She delivered by LSCS. Indication for LSCS was primi with fetal

distress. She delivered a 1.7 kg baby with APGAR 5/10, 8/10. Baby was admitted in NICU for IUGR. Baby died after 4 ays due to sepsis.

- CASE NO: 289

  In control group, Jasmine, 26 yr old, G2P1L1, belonging to class 4 socioeconomic class, 38 weeks GA on admission with AFI 7. Her NST was non reactive, and she went in for labour spontaneously and had clear liquor. Baby weight was 2.7kg and APGAR 8/10, 9/10. Baby was admitted NICU 2 days after birth due to peripheral cyanosis and died after 8 days due to shock and TOF with PHT.

SUMMARY

SUMMARY

In this study perinatal outcome with AFI ≤ 5cm is compared with control group.

About 150 cases were studied in each group.

- 88% of study group and 92.6% of control group belong to 20-30 yrs of age.
- In study group, 38% had vaginal delivery and 46% had LSCS delivery with16% having repeat LSCS. In control group,62% had vaginal delivery and -23.3% had LSCS delivery with 14.7% having repeat LSCS.
- The rate of caesarean section for fetal distress was higher in study group (33.3%) compared to control group (11.3%). The difference was found to be significant. (p - <0.05)
- Induction of labour was higher in study group (36%) compared to control group (30%). The difference was not found to be significant.
- Meconium stained liquor in study group was 46% and control group was 26.6%. The difference was significant (p - <0.05)
- APGAR score <7 at 5 mins was 17.3% in study group as against 2% in control group. The difference was found to be significant.
- Babies weighing less than 2 kg were 25.3% in study group and 8% in control group. This difference was found to be significant. (p- <0.05)

- In study group, 67.3% had reactive NST and 32.7% had non-reactive NST. In control group, 83.3% had reactive NST and 16.7% had non-reactive NST. (p- <0.05)

In study group,

- Risk of having APGAR score <7 at 5 mins was 32.7% nonreactive NST as compared to 9.9% in reactive NST.
- Risk of NICU admission was high in nonreactive NST (83.7%) as compared to reactive NST (36.6%).
- Neonatal death rate was 16.3% in nonreactive NST and 1% in reactive NST. The difference was found to be significant. (p- <0.05)

In control group,

- Risk of having APGAR score <7 at 5 mins, was 8% in nonreactive NST as compared to 0.8% in reactive NST.
- Risk of NICU admission was high in nonreactive NST (44%) as compared to reactive NST (12.8%).
- Neonatal death rate was 4% in nonreactive NST.

CONCLUSION

CONCLUSION

- Oligohydramnios is being detected more often these days due to routinely performed obstetric USG.
- Oligohydramnios is one of the indicators of poor perinatal outcome.
- It is associated with fetal heart rate abnormalities, meconium staining of amniotic fluid, umbilical cord compression, poor tolerance of labour, low APGAR score of fetal acidosis.
- Pregnancy induced hypertension, postdated pregnancies are the commonest causes of reduced amniotic fluid during third trimester of pregnancy.
- Oligohydramnios with reactive NST is associated with good prognosis.
- Oligohydramnios with nonreactive NST needs careful monitoring and results in early delivery, increased incidence of caesarean delivery for fetal distress, NICU admission, low APGAR score at 5 mins, and neonatal death.
- Mode of delivery depends on severity of oligohydramnios and status of fetal wellbeing.
- Caeserean section is mostly required for cases with anhydramnios with intrapartum fetal heart abnormalities. Babies are relatively more prone for certain complications like intrapartum fetal distress, MAS, and birth asphyxia.

- Oligohydramnios associated with IUGR carries a poor perinatal outcome (increased neonatal death, NICU admission, increased rate of caesarean section for fetal distress, very low birth weight). Hence they need good neonatal care.
- From this study, we conclude that oligohydrmanios is a high risk pregnancy and proper antepartum care, intensive fetal surveillance and intrapartum care are required in patient with oligohydramnios.
- Every case of oligohydramnios needs careful antenatal evaluation, parental counseling, individualization, decisions regarding time and mode of delivery.
- Continuous intrapartum fetal monitoring and good neonatal care are necessary for better perinatal outcome.

ANNEXURES

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LIST OF ABBREVIATIONS USED

ACE inhibitors – Angiotensin converting enzyme inhibitors

AFI – Amniotic Fluid Index

AFV – Amniotic fluid volume

CPD – Cephalopelvic disproportion

FH – Fetal heart

GA – Gestational age

IUGR – Intrauterine growth retardation

LN-Labour natural

LSCS-Lower segment caesaerean section

MSAF – Meconium stained amniotic fluid

NICU – Neonatal Intensive Care unit

NST – Non stress test

MAS-Meconium aspiration syndrome

NNEC-NonNecrotizing entero colitis

RDS-Respiratory distress syndrome

PROFORMA

NAME: AGE: IP NO:

LMP: EDD:

ADDRESS:

CONTACT NO:

SOCIOECONOMIC STATUS: Class I/ II/ III/ IV/ V

EDUCATIONAL STATUS:

OBSTETRIC CODE: Primi/ G P L A

GESTATIONAL AGE:

MENSTRUAL CYCLES: Regular/ Irregular

MARITAL HISTORY: Married since

CONSANGUINITY: Consanguinous/ nonconsanguinous

PAST HISTORY:

ASSOCIATED RISK FACTORS: GDM/ PIH/ Breech/ prev LSCS/ postdated

FAMILY HISTORY: Mother/ Father/ Both/ None

If yes, specify condition: DM/ HT/ TB/ BA

GENERAL EXAMINATION: Anemia/ pedal edema

PR: CVS:

BP: RS:

OBSTETRIC EXAMINATION:

FUNDAL HEIGHT:

PRESENTATION:

FHR:

LIQUOR CLINICALLY: Adequate/ not adequate

PER VAGINA: Intact membranes/ ruptured membranes

INVESTIGATIONS:

URINE ALBUMIN:

SUGAR:

DEPOSITS:

HEMOGLOBIN:

BLOOD GROUP:

BLOOD SUGAR:

UREA:

SERUM CREATININE:

ULTRASOUND:

GESTATIONAL AGE:

PLACENTA:

FH:

AFI:

NST: Reactive/ nonreactive

MODE OF DELIVERY:

SPONTANEOUS: INDUCTION:

VAGINAL: INSTRUMENTAL:

LSCS: EMERGENCY/ ELECTIVE

INDICATION FOR LSCS:

INDUCTION DELIVERY INTERVAL:

OXYTOCIN DRIP: Yes/ No

FHR VARIATION: Yes/ No

DURATION OF LABOUR:

RUPTURE OF MEMBRANE:

COLOUR OF LIQUOR: Clear/ thin meconium/ thick meconium

If meconium stained liquor amnioinfusion: yes/no

BABY DETAILS:

CRIED AFTER BIRTH: Yes/ No

SEX:

BIRTH WEIGHT:

APGAR SCORE: 1 MIN:

5 MIN:

BABY ADMISSION IN NICU: Yes/ No

ASSOCIATED COMPLICATION IN BABY:

FOLLOW UP:

CONSENT FORM

DR. ……. has explained to me in my own language that a study on Amniotic fluid volume is being conducted at GTMCH, Theni. I understand that I will undergo Ultrasonographic screening of AFV at my admission and AFV will be used to manage my progress in labour. All other interventions will be as per hospital protocols/ as deemed necessary by the labour room staff. I give my voluntary consent to participate in this study after having given sufficient clarification. I also understand that refusal to participate in this study will not affect the routine care that I receive in this hospital.

Patient's signature:

Patient's name:

Date:

IP no:

MASTER CHART

Sl. No	Name	Age	IP No	Socioeconomic status	Obstetric code	Gestational Age in weeks	High risk	AFI	NST	Onset of labour	Colour of liquor	Mode of delivery	Indication for LSCS	Baby weight	APGAR	NICU admission	Final outcome
1	Maria	29	26215	IV	PRIMI	37	PE	5	NR	I	c	LN		1.9	6	yes	IUGR
2	Subathra	21	27093	III	PRIMI	37		5	R	I	thin	LN		2.6	8	no	
3	Sankari	22	27147	IV	MULTI	38	PL	5	R	S	c	Rpt	IUGR	1.1	9	no	
4	Renuka	32	27065	IV	MULTI	37		4	R	S	thick	LN		2.7	8	yes	
5	Thenmozhi	23	26904	III	PRIMI	41	PD	5	NR	I	thin	LN		1.8	9	no	IUGR
6	Selvi	27	27087	IV	MULTI	38		5	R	S	thick	LN		2.9	8	yes	
7	Mariyammal	25	26925	III	PRIMI	37	PE	5	NR	S	c	LSCS	FD	1.9	8	yes	
8	Vivetha	18	27325	IV	PRIMI	37		3	NR	S	thick	LSCS	FD	2.8	5	yes	
9	Pavithra	24	27674	IV	MULTI	38	PL	4	R	S	c	Rpt	IUGR	1.7	8	no	
10	Meera	21	27802	V	PRIMI	38		4	R	I	thick	LSCS	FI	3.6	8	yes	
11	Prabha	26	28206	III	PRIMI	41	PD	4	R	S	thin	LN		2.6	8	no	
12	Malathi	23	29217	IV	MULTI	37		3	R	S	c	LN		2.7	8	no	
13	Shanthi	20	28183	IV	MULTI	37	PE	5	NR	S	c	LN		1.8	8	yes	IUGR
14	Priya	21	29370	V	MULTI	38	PL	5	R	S	thin	Rpt	CPD	3.4	6	no	
15	Malliga	26	29489	IV	PRIMI	38		4	R	S	c	LN		2.4	9	no	
16	Pandiselvi	24	29968	III	PRIMI	41	PD	4	NR	S	thick	LSCS	FD	1.6	8	yes	Death (IUGR)
17	Lakshmi	23	29907	III	PRIMI	37		5	NR	S	c	LN		2.7	8	no	
18	Amudha	21	30229	IV	MULTI	37	PL	4	R	S	c	Rpt	CPD	3.1	8	no	
19	Kokila	21	30359	IV	PRIMI	38	PE	5	R	S	c	LN		1.6	8	yes	IUGR
20	Chitra	20	30497	IV	MULTI	37		5	R	I	thick	LSCS	CPD	2.8	8	yes	
21	Menaka	25	30638	V	PRIMI	38	B	5	R	S	c	LN		2.9	8	no	
22	Geetha	23	30409	III	PRIMI	41	PD	4	NR	I	thick	LSCS	IUGR	1.8	6	yes	Death (IUGR)
23	Mangai	27	30854	III	PRIMI	37	PE	4	NR	I	c	LSCS	FD	1.9	6	yes	Death (IUGR)
24	Pandiswari	25	29865	III	MULTI	37	PL	3	R	S	c	Rpt	CPD	2.7	8	yes	
25	Kalaiarasi	24	31610	IV	MULTI	41	PD	5	R	I	c	LN		2.7	8	no	
26	Murugeswari	23	31166	IV	MULTI	37		3	NR	I	thick	LSCS	FD	3.1	6	yes	
27	Mahalakshmi	21	30270	V	MULTI	38	PL	3	R	S	thin	Rpt	IUGR	1.8	8	yes	
28	Soniya	26	31678	IV	PRIMI	37	PE	3	R	I	c	LSCS	FD	2.4	8	yes	
29	Sadaiyammal	25	32001	III	PRIMI	38		5	R	I	thin	LSCS	FD	2.6	8	yes	
30	Nandini	24	31907	IV	MULTI	38	PL	3	R	S	c	Rpt	CPD	2.9	5	no	

PE-PRE ECLAMPSIA PL-PREVIOUS LSCS PD-POST DATED FI-FAILED INDUCTION B-BREECH FD-FETAL DISTRESS AFI-AMNIOTIC FLUID INDEX
NST-NON STRESS TEST R-REACTIVE NR-NON REACTIVE I-INDUCED LABOUR S-SPONTANEOUS LABOUR C-CLEAR LIQUOR

31	Tamilselvi	22	32176	III	PRIMI	41	PD	5	NR	S	thick	LSCS	FD	2.5	8	yes	
32	Poovilarani	21	32406	IV	MULTI	37		3	NR	I	thin	LSCS	FD	2.8	9	yes	
33	Srikala	19	32593	V	MULTI	38	PL	4	R	I	c	LSCS	others	2.6	9	no	
34	Vinotha	29	32977	III	PRIMI	38	PE	5	NR	I	thick	LSCS	FD	2.7	8	yes	
35	Vinodhini	27	33298	IV	MULTI	38		4	R	S	thin	LN		2.8	8	no	
36	Suganya	24	33693	IV	MULTI	37	PL	4	R	S	thin	Rpt	CPD	2.4	6	no	
37	Veerasudha	23	33445	III	PRIMI	38		4	NR	I	thick	LSCS	FD	2.9	8	yes	
38	Poomani	22	33790	IV	PRIMI	41	PD	4	R	I	thick	LSCS	FI	2.8	8	yes	
39	Malliga	21	33964	IV	PRIMI	37	PE	5	NR	I	thin	LN		1.6	8	yes	Death (IUGR)
40	Muneeswari	24	33485	III	MULTI	38		3	NR	S	thick	LSCS	FD	2.7	8	yes	
41	Divya	26	33997	III	PRIMI	38		4	NR	S	thick	LSCS	FD	2.5	8	yes	
42	Nagajothi	23	34067	IV	MULTI	37	PL	5	NR	S	thin	Rpt	FD	2.6	5	yes	
43	Sindhuja	21	34177	V	PRIMI	41	PD	2	NR	I	thick	LSCS	FD	2.6	8	yes	
44	Chitra	21	34244	IV	PRIMI	38		4	R	S	c	LN		1.4	7	yes	Death (IUGR)
45	Anandhi	24	34321	III	PRIMI	40	PE	5	R	I	c	LN		2.7	6	no	
46	Hemasudha	26	34368	IV	MULTI	39	PL	2	R	S	thin	Rpt	CPD	2.5	8	no	
47	Meena	25	35217	IV	MULTI	38		4	R	S	thin	LN		2.6	8	no	
48	Chitrakala	27	44319	IV	PRIMI	41	PD	4	R	I	c	LN		2.7	8	no	
49	Ambiga	23	43599	III	PRIMI	38	B	3	NR	S	thick	LSCS	FD	1.7	8	yes	Death (IUGR)
50	Nithya	19	44343	V	MULTI	37		5	R	S	thin	LN		2.8	8	no	
51	Abinaya	29	44341	IV	MULTI	37	PL	3	R	S	c	Rpt	CPD	2.4	8	no	
52	Ferliz	28	44007	IV	PRIMI	38		4	R	I	c	LN		2.8	8	no	
53	Soniya	26	44709	IV	PRIMI	37	PE	4	NR	I	thin	LSCS	FD	1.7	9	yes	IUGR
54	Kousalya	30	44819	III	PRIMI	38		5	R	S	c	LN		2.9	8	no	
55	Mahalakshmi	27	45156	V	PRIMI	41	PD	3	NR	I	thick	LSCS	FD	2.9	8	yes	
56	Devi	25	45662	III	PRIMI	38		3	R	S	c	LN		1.9	8	yes	IUGR
57	Nagashanthi	21	45682	III	MULTI	38		3	R	S	c	LN		2.7	8	no	
58	Chinnathai	32	45606	III	MULTI	38		4	NR	I	c	LN		2.5	6	no	
59	Amudha	23	45746	V	PRIMI	38	PE	3	R	I	c	LSCS	FI	2.8	8	no	
60	Arifa	26	45054	IV	MULTI	38		4	R	S	thick	LSCS	CPD	2.6	8	yes	
61	Alagu	22	46028	IV	MULTI	38	B	5	R	S	c	LSCS	FD	2.6	8	yes	
62	Nadhiya	19	46119	IV	PRIMI	41	PD	3	R	I	thin	LSCS	FI	3.5	8	no	
63	Raasi	24	46518	III	MULTI	37	PL	4	R	S	c	Rpt	FD	2.9	9	yes	
64	Jothimani	21	46244	III	PRIMI	37		5	R	S	thin	LSCS	IUGR	1.5	8	no	
65	Subathra	28	46475	V	PRIMI	38	PE	5	NR	I	c	LSCS	FD	1.8	3	yes	Death (IUGR)
66	Jeyachitra	27	46322	V	MULTI	37	PL	5	NR	S	thick	Rpt	FD	2.9	6	yes	
67	Panchavarnam	27	47143	V	MULTI	37		5	R	I	c	LN		2.8	7	no	
68	Divya	25	47038	IV	PRIMI	41	PD	5	R	S	thin	LN		2.4	6	no	IUGR
69	Saranya	26	47276	IV	MULTI	37	PL	3	R	S	c	Rpt	FD	2.8	8	no	
70	Kanchana	25	47612	III	PRIMI	38		4	R	I	c	LN		2.5	8	no	

71	Selvi	19	47733	III	PRIMI	37	PE	4	R	I	thin	LSCS	FI	1.8	3	yes	IUGR
72	Easwari	28	47741	III	MULTI	37		3	R	S	c	LN		2.9	8	no	
73	Deepa	23	47960	III	PRIMI	38	B	4	NR	S	c	LSCS	FD	2.7	8	no	
74	Selvi	21	48127	III	PRIMI	42	PD	5	R	I	thick	LSCS	FI	2.6	8	yes	
75	Rani	22	47875	III	MULTI	37	PL	3	NR	S	c	Rpt	FD	1.7	6	yes	IUGR
76	Pavithra	29	47448	IV	PRIMI	37	PE	4	R	I	thin	LSCS	FI	1.9	9	yes	IUGR
77	Rajeswari	27	47410	IV	MULTI	37		3	R	S	c	LN		2.8	8	no	
78	Suganthi	25	47421	IV	MULTI	41	PD	4	R	S	thin	LN		2.5	8	no	
79	Palaniyammal	34	47422	IV	MULTI	37	PL	3	NR	S	thick	Rpt	FD	2.7	8	yes	
80	Pandimeena	24	47432	IV	PRIMI	38	PE	3	R	I	c	LSCS	FI	1.9	8	no	IUGR
81	Radhika	23	47438	IV	MULTI	37		3	R	S	c	LN		2.6	8	no	
82	Manimala	18	47440	IV	MULTI	37	PL	5	NR	S	thick	Rpt	FD	2.8	8	yes	
83	Mary	26	47445	V	PRIMI	38		3	NR	S	thick	LSCS	FD	3.2	6	yes	
84	Sumathi	21	47449	V	PRIMI	41	PD	4	R	S	c	LN		2.9	8	no	
85	Janani	23	47504	IV	MULTI	37		5	R	S	c	LN		2.8	8	no	
86	Priya	21	47508	III	PRIMI	38	PE	5	NR	S	thin	LN		1.7	3	yes	Death (IUGR)
87	Vinitha	24	47510	IV	PRIMI	38		5	NR	S	thick	LSCS	FD	2.9	8	yes	
88	Karthika	19	47512	IV	PRIMI	38	B	5	NR	S	c	LSCS	FD	1.8	8	yes	IUGR
89	Nithya	26	47515	III	MULTI	37	PL	3	NR	S	c	Rpt	FD	2.7	9	yes	
90	Kousalya	24	47517	III	MULTI	38		4	R	S	thin	LN		1.9	8	no	IUGR
91	Jeyakodi	29	47526	V	MULTI	39	PL, B	4	R	S	c	Rpt	Breech	2.5	8	no	
92	Anbarasi	26	47539	V	PRIMI	41	PD	3	R	I	thin	LSCS	FI	3.4	8	no	
93	Vinitha	21	47655	III	PRIMI	37		4	R	I	c	LN		1.9	8	yes	IUGR
94	Sahana	19	47680	V	PRIMI	39	PE	5	R	I	thick	LSCS	IUGR	1.6	8	yes	IUGR
95	Fathima	26	47674	III	MULTI	37		5	R	S	c	LN		2.7	9	no	
96	Uma	25	47665	IV	PRIMI	40	B	3	R	S	c	LSCS	Breech	2.4	8	yes	
97	Devi	24	47671	III	MULTI	38	PL	3	NR	S	thick	Rpt	FD	2.8	8	no	
98	Maheswari	23	47669	III	MULTI	37		3	R	I	thick	LSCS	FD	2.7	8	yes	
99	Nivetha	22	47683	III	PRIMI	41	PD	4	NR	I	thick	LSCS	FD	2.5	8	yes	
100	Poongodi	22	22060	IV	PRIMI	38		3	R	S	c	LN		2.6	8	no	
101	Bagylaxmi	33	22038	IV	PRIMI	39	PE, B	4	R	I	c	LSCS	Breech	2.8	8	no	
102	Nithya	28	22093	III	PRIMI	38		3	NR	S	c	LN		2.7	8	no	
103	Ramya	18	22828	V	MULTI	39	B	4	R	S	thin	LN		1.9	6	yes	IUGR
104	Angalaeaswari	23	22185	V	PRIMI	41	PD	5	R	S	thick	LSCS	FD	3.6	8	yes	
105	Alagumeena	22	22687	III	PRIMI	37		4	R	I	c	LN		2.7	8	no	
106	Vinodini	21	22781	IV	MULTI	38	PL, B	5	R	S	c	Rpt	Breech	2.4	9	no	
107	Krishnaveni	26	22897	IV	PRIMI	38	PE	4	NR	S	c	LSCS	FD	1.8	3	yes	IUGR
108	Amutha	25	22898	III	MULTI	40	PL	5	R	S	c	Rpt	FD	2.8	8	yes	
109	Mariyammal	22	23139	IV	PRIMI	41	PD	4	R	I	thick	LN		2.7	8	yes	
110	Bhuvana	21	23247	III	MULTI	39	PE	4	R	S	c	LSCS	IUGR	1.8	8	yes	IUGR

111	Anitha	23	22665	V	PRIMI	39		4	R	S	c	LN		2.5	8	no	
112	Nachiammal	24	23023	V	PRIMI	40		3	R	S	c	LN		2.8	8	no	
113	Anitha	26	23579	IV	MULTI	39		4	R	I	c	LN		2.6	8	no	
114	Alagammal	24	23702	IV	PRIMI	41	PD	5	R	S	thin	LSCS	FD	2.8	8	no	
115	Ajitha	21	23921	IV	MULTI	39		4	R	I	c	LN		2.9	8	no	
116	Veeralakshmi	23	24063	IV	PRIMI	40	B	3	R	S	c	LSCS	Breech	2.5	8	no	
117	Sophiya	23	24281	III	PRIMI	39	PE	4	R	I	thin	LSCS	FI	1.7	3	yes	IUGR
118	Shanthi	21	24705	IV	MULTI	38	PL	3	R	S	c	Rpt	FD	2.4	9	yes	
119	Chinnavalathi	19	23224	IV	PRIMI	37	B	4	R	I	c	LSCS	Breech	2.7	8	no	
120	Meenatchi	22	24819	III	PRIMI	42	PD	3	NR	I	thick	LSCS	FD	2.8	8	no	
121	Selvi	28	24766	III	MULTI	40		4	R	S	c	LN		2.9	8	no	
122	Chellam	26	24859	V	PRIMI	38	B	3	R	S	c	LSCS	Breech	2.7	8	yes	
123	Kaveri	22	24909	III	MULTI	39		3	NR	I	thick	LSCS	FD	3.2	9	yes	
124	Selvamani	21	24966	III	PRIMI	39	PE	4	R	I	c	LSCS	FI	1.9	8	no	
125	Rasiya	24	25407	IV	PRIMI	38	B	5	R	S	c	LSCS	Breech	2.6	8	no	
126	Renuga	22	25393	III	PRIMI	38		3	R	S	c	LN		2.7	8	no	
127	Nandini	21	25549	V	MULTI	41	PD	4	R	I	thick	LSCS	FD	2.6	8	yes	
128	vanitha	23	26029	III	G2P1L1	39	B	3	R	S	c	LN		2.4	8	no	
129	Gowri	24	26289	IV	G2P1L1	37	PE	4	R	I	thin	LSCS	IUGR	1.8	8	yes	IUGR
130	Pavithra	19	26630	IV	PRIMI	38		3	NR	S	thick	LSCS	FD	2.6	8	yes	
131	Mareeswari	33	26840	IV	MULTI	37		4	NR	I	thick	LSCS	FD	2.7	8	yes	
132	Suganya	22	27080	IV	MULTI	37	PL	5	NR	S	thick	Rpt	FD	2.6	3	yes	
133	Poongodi	27	27663	III	MULTI	38		4	R	S	c	LSCS	IUGR	1.6	8	yes	IUGR
134	Jesima	25	27501	III	PRIMI	41	PD	3	R	S	thin	LSCS	IUGR	1.9	8	yes	IUGR
135	Irulayee	26	28969	IV	PRIMI	37	PE	4	R	I	c	LSCS	FI	2.5	8	no	
136	Muthupriya	21	23260	IV	PRIMI	38		5	R	I	c	LSCS	CPD	2.9	8	no	
137	Kanimozhi	31	29091	III	MULTI	37		4	NR	S	c	LN		2.5	8	no	IUGR
138	Deepa	29	29260	IV	PRIMI	41	PD	5	R	I	c	LSCS	FI	3.7	8	no	
139	Latha	26	29036	IV	PRIMI	37	PE	4	NR	S	c	LSCS	FD	1.9	6	yes	IUGR
140	Nandini	18	28652	III	PRIMI	38		3	NR	S	thick	LSCS	FD	3.1	8	yes	
141	Shobana	24	28921	V	PRIMI	37	B	4	R	S	c	LSCS	Breech	2.6	8	no	
142	Jayasurya	25	28930	V	MULTI	37		5	R	S	c	LN		2.4	8	no	IUGR
143	Valarmathi	21	28935	V	PRIMI	41	PD	4	NR	S	thin	LSCS	FD	3.6	8	yes	
144	Sharmila	23	28941	IV	PRIMI	37		3	R	S	thick	LN		2.8	8	yes	
145	Priyanka	22	28947	III	MULTI	37	PL	4	R	S	c	LSCS	others	1.8	8	yes	IUGR
146	Raghumathi	27	28952	III	MULTI	41	PD	3	R	S	c	LN		2.7	9	no	
147	Rani	26	28968	IV	PRIMI	37	B	3	NR	S	thick	LSCS	FD	1.7	8	yes	Death
148	Muthumani	24	28970	III	MULTI	41	PD	4	R	S	c	LN		2.5	8	yes	
149	Jayanthi	25	28985	V	PRIMI	37	PE	4	NR	S	c	LSCS	FD	3.4	5	yes	
150	Nithya	22	28990	III	MULTI	37	PE	5	R	S	c	LN		2.4	8	no	

MASTER CHART

Sl. No	Name	Age	IP No	Socioeconomic status	Obstetric code	Gestational Age in weeks	High risk	AFI	NST	Onset of labour	Colour of liquor	Mode of delivery	Indication for LSCS	Baby weight	APGAR	NICU admission	Final outcome
151	Renuga	19	23115	III	PRIMI	37		8	R	S	thin	LN		3.9	8	no	
152	Kavya	22	23155	IV	MULTI	38	PL, B	8	R	S	c	Rpt	breech	3.4	8	no	
153	Kousalya	24	23186	IV	MULTI	38		8	R	S	c	LN		2.1	9	no	
154	lakshmi	23	23202	V	PRIMI	38	PE	8	NR	I	thick	LSCS	FD	3.7	8	yes	
155	Nirmala	18	23512	III	MULTI	38		9	R	S	thin	LN		2.4	8	no	
156	Latha	26	23545	III	PRIMI	37		7	NR	S	c	LN		3.7	8	no	
157	Aandal	27	23590	III	PRIMI	41	PD	9	R	I	thick	LSCS	FD	2.4	8	no	
158	Aarthi	28	23650	IV	MULTI	38		10	R	S	c	LN		2.3	9	no	
159	Deepa	29	23690	III	MULTI	39		9	R	S	c	LN		2.4	8	no	
160	Hema	19	23712	V	PRIMI	41	PD	9	R	I	thick	LSCS	FD	2.3	8	yes	
161	Jasmine	27	23748	V	PRIMI	38		8	R	S	c	LN		2.1	8	no	
162	Kripa	26	23795	IV	MULTI	38	PL	10	R	S	c	Rpt	others	3.6	9	no	
163	Alagammanl	25	23820	IV	PRIMI	38		8	NR	S	thin	LN		2.3	8	no	
164	Angulakshmi	18	23863	IV	PRIMI	39	PE	9	NR	I	thick	LSCS	FD	3.6	8	yes	
165	Chitra	29	23894	V	MULTI	39		9	R	S	c	LN		3.6	8	no	
166	Valarmathi	29	23900	IV	PRIMI	38		8	R	S	thin	LSCS	FD	2.6	8	no	
167	Geetha	28	23901	III	PRIMI	41	PD	8	R	I	c	LN		2.7	8	no	
168	Hemalatha	27	23911	III	MULTI	38		8	NR	S	thin	LN		3.5	8	no	
169	Ganeswari	26	23935	III	PRIMI	38	PE	10	R	I	c	LN		2.8	8	no	
170	Malathi	28	23956	III	MULTI	37		8	NR	S	c	LN		2.7	8	no	
171	Meena	27	23989	III	PRIMI	41	PD	7	NR	I	thick	LSCS	FD	2.3	8	yes	
172	Nalini	30	24005	III	MULTI	38	PL	9	R	S	c	Rpt	CPD	3.4	9	no	
173	Nila	18	24561	IV	MULTI	38		9	R	S	c	LN		2.6	8	no	
174	Miruna	24	24885	IV	PRIMI	41	PD	7	NR	I	c	LN		2.7	9	no	
175	Kalaiarasi	25	24965	IV	MULTI	41	PD	8	R	I	c	LSCS	FI	2.6	8	no	
176	Bala	23	25101	IV	PRIMI	37		8	R	S	thin	LN		2.5	9	no	
177	Ambigai	27	25109	V	MULTI	38	PE	9	NR	I	c	LN		1.9	9	yes	IUGR
178	Gayathri	22	25135	III	PRIMI	38		9	R	S	c	LN		2.7	9	no	
179	Nandini	18	25165	III	MULTI	37	PL	8	R	S	c	Rpt	CPD	3.6	8	no	
180	Malini	21	25187	III	PRIMI	38		9	R	S	thin	LN		2.8	8	no	

PE-PRE ECLAMPSIA PL-PREVIOUS LSCS PD-POST DATED FI-FAILED INDUCTION B-BREECH FD-FETAL DISTRESS AFI-AMNIOTIC FLUID INDEX
NST-NON STRESS TEST R-REACTIVE NR-NON REACTIVE I-INDUCED LABOUR S-SPONTANEOUS LABOUR C-CLEAR LIQUOR

181	Janaki	24	25188	III	MULTI	38	PE	6	NR	I	thick	LN		2.7	8	yes	
182	Jennathul	21	25194	III	PRIMI	37		10	R	S	thin	LN		2.7	8	no	
183	Loganayaki	32	25222	III	MULTI	38	PL	8	NR	S	c	Rpt	CPD	3.7	9	no	
184	Pandiammal	23	25326	III	PRIMI	39		10	R	S	c	LSCS	CPD	3.6	8	no	
185	Veena	27	25445	III	PRIMI	41	PD	10	R	I	c	LN		2.6	8	no	
186	Murugammal	22	25482	III	MULTI	38	PE	10	R	S	c	LSCS	CPD	3.7	9	no	
187	Alagammal	26	25502	IV	PRIMI	37	PE	9	R	I	thin	LN		2.8	8	no	
188	Veni	24	25533	IV	MULTI	38		7	R	S	c	LN		2.9	8	no	
189	Thangam	21	25564	III	PRIMI	39		6	NR	S	c	LSCS	CPD	3.6	9	no	
190	Vaishali	23	25575	III	MULTI	38		8	R	S	c	LN		2.9	8	no	
191	Priya	22	25582	V	PRIMI	39		8	NR	I	thick	LSCS	FD	2.8	9	yes	
192	Sundari	33	25601	III	PRIMI	38		7	R	S	c	LN		3	8	no	
193	Sheela	25	25622	III	MULTI	38		8	R	S	c	LN		1.8	8	no	IUGR
194	Thilagavathy	27	25645	III	PRIMI	38	PE	8	R	I	c	LN		2.9	8	no	
195	Fathima	28	25646	IV	PRIMI	37		8	R	S	thin	LN		3.5	9	no	
196	Latha	27	25679	IV	MULTI	37	PL	9	R	S	c	Rpt	CPD	3.6	8	no	
197	Maya	26	25698	III	PRIMI	38	PE	9	R	I	c	LN		2.7	9	no	
198	Chitra	25	26241	III	MULTI	38		9	R	I	thin	LSCS	FD	1.8	6	yes	IUGR
199	Deepa	24	26249	IV	PRIMI	42	PD	8	R	I	thin	LN		3.5	8	no	
200	Elakiya	23	26458	IV	PRIMI	38		7	R	S	c	LN		2.6	9	no	
201	Firdouse	29	26586	IV	MULTI	37	PL	6	R	S	c	Rpt	CPD	3.7	8	no	
202	Pandiselvi	24	26777	IV	MULTI	38		8	R	S	c	LN		2.4	8	no	
203	Haritha	29	26959	IV	PRIMI	39		8	R	I	c	LSCS	FI	2.7	8	yes	
204	Revathi	28	27101	IV	PRIMI	38		9	R	S	thin	LN		2.9	8	no	
205	Dhanalakshmi	28	27159	IV	MULTI	39		9	R	S	c	LN		3	9	no	
206	Sangeetha	26	27168	IV	PRIMI	39		8	NR	I	thick	LSCS	FD	3.6	6	no	
207	madhu	36	27248	III	MULTI	38		8	R	S	c	LSCS	IUGR	1.8	8	no	IUGR
208	Renuka	24	27345	IV	PRIMI	38		7	R	S	c	LN		2.7	9	no	
209	Wahitha	21	27358	IV	MULTI	38	PL	7	R	S	c	Rpt	CPD	3.6	8	no	
210	Chitra	25	27568	IV	PRIMI	38	PE	7	R	S	thin	LN		2.8	8	yes	
211	Seetha	26	27598	III	PRIMI	39		6	R	S	thin	LN		2.7	8	no	
212	Sathya	23	27869	IV	PRIMI	38		9	R	S	c	LN		2.6	8	no	
213	Gayathri	21	28125	IV	MULTI	37	PL	9	R	S	c	Rpt	CPD	3.4	9	no	
214	Amutha	24	28165	III	PRIMI	39		6	R	I	c	LSCS	FI	2.7	8	yes	
215	Janaki	21	28454	V	MULTI	38	PE	9	R	S	c	LN		2.8	9	no	
216	Kalyani	22	28495	V	PRIMI	39		9	R	S	c	LN		2.6	8	no	
217	Bala	34	28678	IV	MULTI	38	PL	6	R	S	c	Rpt	CPD	3.4	8	no	
218	Beena	28	28789	IV	PRIMI	39		9	R	S	c	LN		2.4	9	no	
219	Vinothini	30	28790	III	PRIMI	38	PE	8	R	S	thin	LN		2.8	9	no	
220	Lokiya	29	28945	III	MULTI	39		8	R	S	c	LN		2.7	8	no	

221	Kanniga	27	29903	IV	PRIMI	38		8	R	I	c	LSCS	FI	2.6	8	no	
222	Karthiga	21	29154	III	MULTI	38	PL	7	NR	S	c	Rpt	CPD	2.4	9	no	
223	Muniyammal	24	29465	IV	MULTI	39		8	R	S	c	LN		2.7	8	no	
224	Mala	22	29868	IV	PRIMI	38		7	NR	I	thick	LSCS	FD	2.7	8	yes	
225	Varshini	25	29968	III	PRIMI	37		10	R	S	c	LN		2.8	9	no	
226	Nivetha	25	30005	III	PRIMI	37	PE	8	R	I	thin	LN		2.7	8	yes	
227	Chellamal	24	30045	IV	PRIMI	38		8	R	S	c	LN		2.4	8	no	
228	Sivaranjini	19	30090	IV	MULTI	38	PL	8	R	S	c	Rpt	IUGR	1.6	9	no	IUGR
229	Vigneswari	23	30124	V	PRIMI	39		9	R	S	thin	LN		2.9	8	no	
230	Saradha	21	30265	V	MULTI	41	PD	7	NR	I	c	LSCS	FD	2.9	8	yes	
231	Pradeepa	24	30285	III	PRIMI	39		6	R	S	c	LN		2.8	9	no	
232	Krithiga	22	30298	IV	PRIMI	38		9	R	I	c	LSCS	FI	2.5	8	no	
233	Lavanya	28	30356	III	MULTI	39		7	R	S	thin	LN		2.8	8	no	
234	Logeswari	30	30385	IV	MULTI	38		8	R	S	c	LSCS	CPD	3.4	9	no	
235	Priya	29	30454	III	PRIMI	39		8	NR	S	c	LN		2.8	8	no	
236	Malliga	35	30488	V	MULTI	38		8	R	I	c	LSCS	FI	2.7	8	no	
237	Meena	27	30495	III	PRIMI	40		8	R	S	c	LN		2.8	9	no	
238	Nalini	25	31000	III	PRIMI	39		9	R	S	thin	LN		2.7	8	no	
239	Radhika	24	31025	III	MULTI	38	PL	9	R	S	c	Rpt	CPD	3.5	9	no	
240	Ragini	22	31065	III	PRIMI	39	PE	8	R	I	c	LN		2.9	8	yes	
241	Swetha	27	31098	IV	PRIMI	41	PD	7	R	I	thin	LN		2.8	8	yes	
242	Shalini	28	31110	IV	MULTI	38	PL	9	R	S	c	Rpt	IUGR	1.7	8	no	IUGR
243	Menaka	22	31133	IV	PRIMI	38	PE	7	R	I	c	LN		2.9	9	no	
244	Kavitha	24	31142	IV	MULTI	39		8	R	S	c	LN		2.3	8	no	
245	Kanimozhi	23	31156	IV	PRIMI	39		7	NR	I	thick	LSCS	FD	1.8	8	yes	IUGR
246	Jamila	23	31177	IV	PRIMI	38	PE	7	R	I	thin	LN		2.6	8	no	
247	Suruliyammal	21	32232	III	MULTI	38		7	R	S	c	LSCS	CPD	2.7	9	no	
248	Pandeeswari	26	32356	IV	PRIMI	41	PD	6	NR	I	c	LN		2.4	9	no	
249	Vanitha	25	32656	V	MULTI	38		6	NR	S	c	LSCS	FD	2.8	8	yes	
250	Chellam	24	32785	III	PRIMI	39		8	R	S	c	LN		2.9	8	no	
251	Solaiammal	22	32959	V	PRIMI	38	PE	9	R	I	thin	LN		2.6	9	no	
252	Lavanya	21	33045	III	MULTI	37		9	R	S	c	LN		2.4	8	no	
253	Anusya	23	33525	III	PRIMI	39		7	NR	S	thick	LSCS	FD	1.8	6	yes	IUGR
254	Sangeetha	24	33626	III	PRIMI	38		8	R	S	c	LN		2.4	8	no	
255	Dhanalakshmi	25	33754	IV	MULTI	39		8	R	S	c	LN		3	9	no	
256	Nagammal	27	34521	III	PRIMI	38	PE	8	R	I	thin	LN		2.3	8	no	
257	Rani	28	34582	IV	MULTI	38	PL	8	R	S	c	Rpt	IUGR	1.8	9	no	IUGR
258	Parvathi	21	34658	III	MULTI	38		8	R	S	c	LN		2.3	8	no	
259	Harini	23	34999	IV	PRIMI	39	PE	9	R	I	thin	LN		1.8	8	yes	
260	Gayathri	29	35011	III	PRIMI	41	PD	8	R	I	thin	LN		2.4	9	no	

261	Leela	26	35056	IV	MULTI	39		9	R	S	c	LN		2.9	9	no	
262	Chitra	25	35074	III	PRIMI	38		6	NR	I	c	LSCS	FI	2.8	8	no	
263	Maria	24	35085	IV	PRIMI	39		8	R	S	c	LN		2.7	8	no	
264	Nithya	22	35102	IV	MULTI	38	PL	9	R	S	c	Rpt	CPD	3.1	9	no	
265	Rajeswari	21	35132	V	PRIMI	39		9	R	S	c	LN		2.4	8	no	
266	Thulasi	23	35144	IV	PRIMI	38		9	R	S	c	LN		2.5	9	no	
267	Ambigai	21	35156	IV	MULTI	38	PL,B	7	R	S	c	Rpt	breech	2.8	8	no	
268	Janani	29	35285	IV	PRIMI	38		8	R	S	c	LN		2.7	9	no	
269	Maya	27	35365	III	MULTI	37		7	R	S	c	LN		2.6	9	no	
270	Bharathi	26	35348	III	PRIMI	39		7	R	S	c	LN		1.9	8	no	IUGR
271	Muniammal	28	35377	III	MULTI	38		8	R	S	c	LN		2.8	8	no	
272	Thilagavathy	27	35956	III	PRIMI	39		9	R	S	c	LN		2.7	9	no	
273	Gayathri	24	36420	III	PRIMI	38		8	R	S	c	LN		2.2	8	no	
274	Janaki	25	36865	III	MULTI	37	PL	9	R	S	c	Rpt	CPD	3.5	9	no	
275	Kalyani	26	36965	IV	PRIMI	38		9	R	S	c	LN		2.8	9	no	
276	Parvathi	23	36011	IV	MULTI	38		9	R	I	c	LSCS	FI	3.6	8	yes	
277	Kalpana	21	36025	IV	PRIMI	37		7	R	S	c	LN		2.9	8	no	
278	Seetha	21	36092	IV	MULTI	38		9	R	S	c	LN		2.7	8	no	
279	Yamini	23	36102	III	PRIMI	39		9	R	S	c	LN		2.6	9	no	
280	Suganya	24	36145	III	MULTI	38	PL	7	R	S	c	Rpt	CPD	2.3	9	no	
281	Revathi	26	36258	IV	PRIMI	39		8	R	S	c	LN		2.7	8	no	
282	Maheswari	28	36956	III	MULTI	39		8	R	S	c	LN		2.7	9	no	
283	Mareeswari	29	37562	IV	PRIMI	39		7	R	I	thin	LSCS	FI	3.6	9	yes	
284	Santhanammal	21	37865	III	PRIMI	38		9	R	S	c	LN		2.3	8	no	
285	Rahini	22	38659	IV	PRIMI	39		8	R	S	c	LN		2.7	9	no	
286	Buvana	29	38958	III	PRIMI	38	PE	7	R	I	c	LN		2.8	8	yes	
287	Subbulakshmi	24	39565	IV	MULTI	39		8	R	S	c	LN		2.9	9	no	
288	Pappa	25	39788	IV	PRIMI	38		8	R	I	thin	LSCS	FD	2.9	8	yes	
289	Jasmine	26	40024	IV	MULTI	38		7	NR	S	c	LN		2.7	8	yes	Death
290	Thangam	23	40125	III	PRIMI	39		10	R	I	c	LSCS	FI	2.6	8	no	
291	Pradeepa	21	40456	IV	PRIMI	39		10	R	I	c	LSCS	IUGR	1.9	9	yes	IUGR
292	Sarala	22	40758	III	MULTI	38	PL, B	9	R	S	c	Rpt	breech	2.8	8	no	
293	Radhika	26	41236	III	PRIMI	42	PD	9	R	I	thin	LSCS	FI	2.8	8	yes	
294	Kousalya	24	41586	III	MULTI	38		8	R	S	c	LN		2.9	8	no	
295	Kavitha	22	43569	III	PRIMI	39		7	R	S	c	LSCS	FD	2.8	9	yes	
296	Lavanya	21	43586	III	MULTI	38	PL	7	R	S	c	Rpt	others	2.7	9	no	
297	Sadhana	25	44577	III	MULTI	38		8	R	S	c	LN		2.9	8	no	
298	Gowri	27	45858	IV	PRIMI	38		6	NR	S	c	LSCS	FD	2.8	8	no	
299	Thulasi	29	45966	III	MULTI	38		9	R	S	c	LN		2.7	9	no	
300	Maria	24	46859	III	MULTI	37	PL	8	R	S	c	Rpt	CPD	2.6	8	no	

Ref. No. 2643/ME1/15

Government Theni Medical College
Theni. Dated: 22.06.2015

Institutional Ethical Committee:
Convenor:

Dr. **K. Kathirkamu, M.S.,**
Dean (FAC)
Govt. Theni Medical College
Theni

Sub: Medical Education – Govt. Theni Medical College, Theni – Ethical Committee – Minutes – Communicated – Reg.

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The Ethical Committee Meeting of the Govt. Theni Medical College, Theni was held at 12.00 noon on 22.06.2015 at Conference Hall, Near Dean's Chamber, Government Theni Medical College, Theni.

The following Members of the Committee have attended the Meeting.

1.	**Convener**	:	**Dr. K. Kathirkamu, M.S.,** **DEAN (FAC)**
2.	**Member Secretary**	:	**Dr. B. Jayakumar, M.S.,** **Deputy Superintendent**
4.	**Members**		
	Professor of Medicine	:	**Dr. P. Purushothaman, M.D.,**
	Professor of Surgery	:	**Dr. R. Murugesan, M.S.,**
	Professor of Obs. & Gynaec.	:	**Dr. Thangamani, M.D., O.G.,**
	Professor of Micro Biology	:	**Dr. K.M. Mythreyee, M.D.,**
5.	**Chairman** **(Private Consultant)**	:	**Dr. Paulraj, M.D.,** Ramya Clinic, Periyakulam Road, Theni.
6.	**Lawyer**	:	**Thiru.K.Murugesan, B.Com., B.L.,** S/o.Kamaraj, Ambedkar Nagar, Varusanadu, Theni District.
7.	**Sociologist**	:	**Sr. Anaestescia** Director, Jeevan Jothi Hospital Community Care Centre, Periyakulam Road, Kailasapatti, Theni Dist.
8.	**Public**	:	**Mr. P. Deenadhayalan, M.A.,** Land Lord, Koduvilarpatti, Theni District.

The following Project was approved by the Committee:

Name of the PG	Course	Name of the Project	Remarks
Dr. Rosalind	II Year - M.S. OG Dept. of Obs. & Gynaec. GTMC, Theni	Perinatal outcome in term oligohydramios.	Approved

Please note that the investigator should adhere the following: He/she should get a detailed informed consent from the Patients/participants and maintain Confidentially.

1. He/she should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution.
2. He/she should inform the Institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. He/she should not deviate for the area of the work for which applied for Ethical Clearance. He/She should inform the Institution Ethical Committee immediately, in case of any adverse events or any serious adverse reactions.
4. He/she should abide to the rules and regulations of the institution.
5. He/she should complete the work within the specific period and apply for if any extension of time is required. He/she should apply for permission again and do the work.
6. He/she should submit the summary of the research work to the Ethical Committee on completion of the work.
7. He/she should not claim any funds from the institution while doing the work or on completion.
8. He/she should understand that the members of Institutional Ethical Committee have the right to monitor the work with prior intimation.

Chairman

Convenor

Dr.M. PALRAJ,M.D.,
Civil Surgeon Retd.,
RAMYA HOSPITAL
574, Periyakulam Road,
THENI - 625 531.
Regn. No: 28094

To

The above PG Student – through Head of the Department concerned.

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IN OLIGOHYDRAMNIOS IN TERM PREGNANCY

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Dissertation submitted to

The Tamilnadu Dr.M.G.R Medical University

in partial fulfillment of the requirement for the award of

MS BRANCH II

OBSTETRICS AND GYNAECOLOGY

THE TAMILNADU

Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

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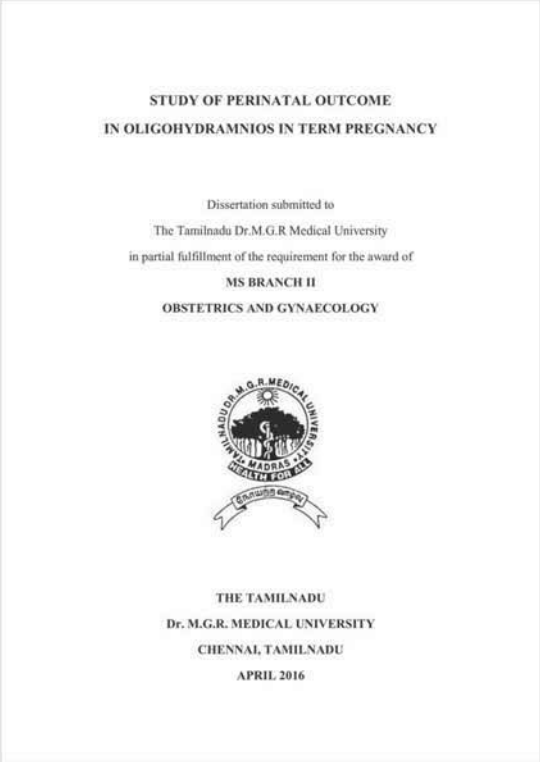

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IN OLIGOHYDRAMNIOS IN TERM PREGNANCY

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